HAZARDOUS WASTE CONTAMINATION OF WATER RESOURCES

(EPA Implementation of the Superfund Program and Lead Pollution Problems in Dallas, TX)

(98-73)

HEARINGS

BEFORE THE

SUBCOMMITTEE ON INVESTIGATIONS AND OVERSIGHT

OF THE

COMMITTEE ON PUBLIC WORKS AND TRANSPORTATION

HOUSE OF REPRESENTATIVES

NINETY-EIGHTH CONGRESS

FIRST SESSION

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CONTENTS

Proceedings of: Page
March 16, 1983 ... 1
March 24, 1983 ... 245

TESTIMONY

Barnes, Dr. Donald, Science Advisor to the Assistant Administrator for Pesticides and Toxic Substances, U.S. Environmental Protection Agency ... 58
Cross, Ms. Lois, Dallas, TX ... 291
Davis, Dr. Allyn M., Director, Air and Waste Management Division, U.S. Environmental Protection Agency, Region VI, Dallas, TX ... 308
Dyer, Norman, Ph.D., Chief, Pesticides and Toxics Branch, U.S. Environmental Protection Agency, Region VI, Dallas, TX ... 308
Frost, Hon. Martin, Congressman from Texas ... 256
Hernandez, John W., Jr., Acting Administrator, U.S. Environmental Protection Agency; accompanied by John E. Daniel, Chief of Staff, U.S. Environmental Protection Agency; and Joseph A. Cannon, Associate Administrator of Policy and Resource Management, U.S. Environmental Protection Agency ... 5
McAnalley, Bill, Ph.D., Toxicologist, Dallas, TX ... 343
McPhee, Jonathan T., Assistant Regional Counsel, Region V, Chicago, IL, U.S. Environmental Protection Agency ... 5
Phillips, Mrs. Frances, Deputy Regional Administrator, U.S. Environmental Protection Agency, Region VI, Dallas, TX ... 308
Saginaw, Ms. Jane, Attorney, Frederick M. Baron and Associates, Dallas, TX.. 291
Spears, Mrs. Patricia, Dallas, TX ... 291
Whittington, Richard, Regional Administrator, U.S. Environmental Protection Agency, Region VI, Dallas, TX ... 308
Wodka, Steven, Director, Health Effects Research, Frederick M. Baron and Associates, Dallas, TX ... 291

MATERIAL RECEIVED FOR THE RECORD

Albosta, Hon. Donald Joseph, Congressman from Michigan; letter dated March 7, 1979, to Douglas Costle, Administrator, U.S. Environmental Protection Agency, regarding emergency suspension order for the herbicide 2, 4, 5-T ... 79
Boll, Thomas J., Ph.D., professor of psychology, pediatrics and neurological surgery and director, medical psychology program, the University of Alabama in Birmingham, Birmingham, AL; neuropsychological evaluation on December 14, 1982, for Demitria Demmings; and Patrick Demings on August 5, 1982 ... 299
Brown, Michael A., Enforcement Counsel, Office of Enforcement Counsel, U.S. Environmental Protection Agency, Washington, DC; letter dated March 25, 1983, to Hon. Elliott H. Levitas, Chairman, Subcommittee on Investigations and Oversight, concerning a question by Congressman Albosta at a February 16 hearing before the full committee ... 199
Brown, Paul H., Cochairman, Chlorinated Dioxins Work Group (TS-777), U.S. Environmental Protection Agency, Washington, DC; memorandum to Karl Bremer, Toxic Substances Coordinator, Region V, U.S. Environmental Protection Agency, regarding Dr. Milt Clark's Dioxin Paper ... 208
Cohan, Robert M., resident attorney, Wald, Harkrader & Ross, Dallas, TX; letter dated September 8, 1981, to Mrs. Frances E. Phillips, Acting Regional Administrator, Dallas Regional Office, Environmental Protection Agency, concerning RSR Corp ... 354

Crosby, Dr. Ernest C. and V.K. Argento, Department of Civil Engineering, the University of Texas at Arlington, Arlington, TX; Environmental Concentrations of Lead and Cadmium in the Areas of Secondary Lead Smelters and a Battery Manufacturing Plant in Dallas, TX, prepared for: Dr. Larry L. Thomas, Toxic Substances Specialist, Air and Hazardous Materials Division, U.S. Environmental Protection Agency, Region VI, Dallas, TX 130

Dallas City Health Department and Dallas Area Respiratory Health Association; a report on a study to determine the blood lead levels in Dallas children 325

Dyer, Norman E., Chief, Pesticides and Toxic Substances Branch, Region VI, U.S. Environmental Protection Agency; letter dated March 29, 1983, to Robert S. Prolman, professional staff member, Subcommittee on Investigations and Oversight, submitting documentation for certain statements made in reply to questions during the hearing of March 24, 1983 432

Fielding, Fred F., Counsel to the President, The White House, Washington, DC; letter dated March 23, 1983, to Hon. Elliott H. Levitas, chairman, Subcommittee on Investigations and Oversight, enclosing Memorandum of Understanding dated March 22, 1983; and letter dated March 21, 1983, from Hon. Elliott H. Levitas to Fred Fielding regarding changes to the Memorandum of Understanding enclosed in letter of March 18, 1983 247

Frost, Hon. Martin, Congressman from Texas:

- Statement 260
- Miscellaneous correspondence between Hon. Martin Frost, U.S. Environmental Protection Agency, and State of Texas officials; concerning lead contamination among the citizens of Dallas, TX 265

Hernandez, John W., Jr., Acting Administrator, U.S. Environmental Protection Agency; statement 8

McAnalley, Bill, Regional Expert in Toxicology; memorandum dated September 4, 1981, to Bill Hathaway, Enforcement Division, U.S. Environmental Protection Agency concerning lead poisoning 365

Phillips, Mrs. Frances, Deputy Regional Administrator, Region VI, U.S. Environmental Protection Agency; letter dated April 6, 1983, to Hon. Elliott H. Levitas, chairman, Subcommittee on Investigations and Oversight, submitting for the record several documents from the regional files that were requested at hearing on March 24, 1983 368

U.S. Environmental Protection Agency:

- Region V, Chicago, IL; a report on Polychorlinated Dibenzo-P-Dioxins (PCDD's) and Polychlorinated Dibenzofurans (PCDF's): A summary of studies conducted in the Great Lakes area, dated July 1981 81
- Environmental News, June 1, 1981; preliminary study, released by Region VI, investigating lead and cadmium levels near industrial sources in Dallas, TX 372
- Memorandum dated June 1, 1981, from Deborah DeBerry-Kershaw, Director, Office of Public Information to Byron Nelson, Press Officer; concerning Dallas lead study press release 375

West, John A., Pesticides and Toxic Substances Branch, Region VI, U.S. Environemntal Protection Agency; memorandum dated April 5, 1983, to Allyn M. Davis, Director, Air & Waste Management Division, U.S. Environmental Protection Agency, regarding speaking points of Frances Phillips in reference to Dallas lead study 431

HAZARDOUS WASTE CONTAMINATION OF WATER RESOURCES

(EPA Implementation of the Superfund Program and Lead Pollution Problems in Dallas, TX)

WEDNESDAY, MARCH 16, 1983

House of Representatives,
Subcommittee on Investigations and Oversight,
Committee on Public Works and Transportation,
Washington, DC.

The subcommittee met, pursuant to notice, at 9:35 a.m., in room 2167, Rayburn House Office Building, Hon. Elliott H. Levitas (chairman of the subcommittee) presiding.

Mr. Levitas. The subcommittee will please come to order.

This morning, the Subcommittee on Investigations and Oversight of the Committee on Public Works and Transportation continues its ongoing inquiry into the matter of hazardous waste contamination of America's water resources and the U.S. Environmental Protection Agency's efforts to carry out the Superfund Program for cleaning up hazardous waste dumps.

Our efforts to date to examine this subject and EPA's efforts to implement this law have raised a number of questions about both the adequacy of the statute and of the EPA to effectively and expeditiously clean up these sites.

Last week's resignation of Mrs. Anne Burford as Administrator of the Environmental Protection Agency has led to the designation of Dr. John Hernandez, formerly Deputy Administrator, as Acting Administrator. And our purpose this morning is to take this opportunity to discuss with him a number of questions pertaining to Agency policy.

I know that there are a number of areas that we will be getting into today, some of which are rather general in nature, pertaining to the ongoing investigation of the Superfund law, which is the primary and continuing thrust of our investigation.

Some of them will relate to what I have described as the jack rabbits that keep jumping up at us every time we walk down the path, which we obviously have to follow until we are able to determine the direction in which they are headed.

So, I am satisfied we will be getting into a lot of different areas and subject matters during the discussion today. Today, we will be hearing from Dr. John W. Hernandez, the acting administrator. He is accompanied by Mr. John E. Daniel, Chief of Staff of the U.S.

Environmental Protection Agency, and Mr. Joseph A. Cannon, Associate Administrator for Policy and Resource Management of the U.S. Environmental Protection Agency.

In addition to that, they are accompanied by others whom I will let them introduce, some of whom have previously testified before this subcommittee.

Dr. Hernandez, I want to welcome you and your associates here this morning. As I am sure you are aware, my concern has been and continues to be the nature of EPA's policies with respect to the environment in general and the Superfund, in particular. I will be looking forward to our discussion today.

At this time, I would like to inquire whether Mr. Molinari, the gentleman from New York, has any opening statement to make.

Mr. **MOLINARI**. Thank you, Mr. Chairman. I have a brief statement.

Mr. Hernandez, this subcommittee appreciates your being here this morning to give testimony. It was my hope a few days ago that at this hearing we would be speaking to you about the future policies and management of the Environmental Protection Agency.

All of us here know how necessary it is that we get on with the work of correcting past policy problems and insuring that hazardous waste laws are vigorously and properly implemented from here on in.

It was my hope that with your assuming the role of acting administrator, there might be a partial clearing of the dark cloud that has hung over EPA for months now. However, in light of the press accounts in the last 24 hours of your dealings with Dow Chemical Co. and changes in a 1981 draft report on dioxin contamination in Michigan, it is clear that the cloud of suspicion still hangs over EPA.

I hope that you can provide satisfactory testimony regarding your role in a Dow incident and let this subcommittee know who was responsible for what actions. As acting administrator, Mr. Hernandez, you, by necessity, will play a critical role in getting EPA onto its feet again and heading in the right direction. It now seems that your ability to do this would be dependent upon your response to the Dow charges.

Mr. Chairman, I think it would be helpful if we could at this point ask Dr. Hernandez to introduce for the edification of the members of the subcommittee the other members of EPA who are present with him who have not been identified at this point.

Dr. **HERNANDEZ**. Mr. Chairman, thank you very much. I am pleased to be here today and to have with me, on my left, Mike Brown, who is head of our enforcement program at EPA, the overall effort.

Mr. **SNYDER**. Excuse me Dr. Hernandez.

Dr. **HERNANDEZ**. Yes.

Mr. **SNYDER**. Might I ask unanimous consent to submit my statement?

Mr. **LEVITAS**. No. I am going to recognize other members who have asked to make opening statements as soon as Dr. Hernandez has introduced the people accompanying him.

Dr. **HERNANDEZ**. Mr. Chairman, on my immediate right hand is Mr. Bill Hedeman, who is head of the so-called Superfund Program at EPA and who has promised he will stop smoking if we get the pressure off him here.

You have met Joe Cannon, who is the next one down, followed by Lou Crampton, who heads our program for regulatory review and—what is it, Lou?

Mr. **Crampton**. Management systems and evaluation.

Dr. **Hernandez**. Management systems and evaluation. They have got a lot of names for this.

Mr. Chairman, back to you.

Mr. **Levitas**. Thank you. I am very pleased to see with us today the chairman of our full committee, Congressman Howard, and also the ranking member of the full committee, Congressman Snyder of Kentucky.

Mr. **Snyder**. Thank you, Mr. Chairman. I want to welcome the panel today; in particular, Dr. Hernandez I want to welcome you in your inaugural appearance before a committee of Congress in your new position as the Actinq Administrator.

Mr. Chairman, among those of us who have worked with Dr. Hernandez over the past 2 years in his prior appearances before the committee and its various subcommittees and in private consultations, his reputation for straight talk and thoughtful discussion of environmental issues, particularly those involved with the Clean Water Act and its related programs, is of the highest regard.

As my colleague from New York has mentioned, recent media reports have raised charges of impropriety in the handling of certain EPA reports dealing with chemical and hazardous waste problems in Michigan. I trust that our hearing today will provide Dr. Hernandez the opportunity to respond and clear the record on that issue.

Pending resolution of that issue, Mr. Chairman, it is my opinion that his technical expertise and professionalism, coupled with a large dose of good, old-fashioned commonsense, have made him a valuable asset to the Agency and to the administration. These same qualities, I trust, will continue to serve him well in the difficult role that the President has asked him to assume.

The subcommittee today continues its investigation and oversight of EPA's administration of the Superfund Program. There remain unanswered many questions which the resignation of Mrs. Burford alone would not and could not resolve. Nevertheless, her departure, whether warranted or not, clears the way for a fresh approach to the conduct of this subcommittee's inquiry into the program to clean up hazardous waste from inactive and abandoned dump sites.

Let us hope that today's hearing is quite literally the beginning of the end. The end which we all seek, of course, is the enactment of adjustments to the Superfund law which may be required in order to expedite the handling of one of this Nation's most pressing environmental problems.

Thank you, Mr. Chairman.

Mr. **Levitas**. Thank you, Mr. Snyder.

Does any other member desire to make an opening statement? Mr. Albosta, the gentleman from Michigan?

Mr. **Albosta**. Mr. Chairman and fellow members of the Subcommittee on Investigations and Oversight, I appreciate the opportunity to say a few words before we hear from Dr. John Hernandez, Acting Administrator of the Environmental Protection Agency, this morning.

In the last several days, a number of extremely serious allegations have come to light concerning possible dioxin pollution in my own State of Michigan involving my district and a major chemical company located there. It has been charged that Dow Chemical Co., headquartered in Midland, MI, has been permitted to review an EPA report on dioxin pollution in Michigan while the report was still being reviewed by EPA personnel in their Washington office.

These are serious charges, Mr. Chairman, that disturb me a great deal. I am concerned about the health of the people in Michigan and about the integrity of EPA, one of the principal agencies responsible for protecting the health of our citizens. We need to determine at today's hearing whether the exchange of information with Dow was proper or improper.

More importantly, we need to determine where dioxin pollution is coming from in Michigan, what health hazards dioxin poses to the people of Michigan, and what needs to be done by industry, the State of Michigan and the Federal Government to effectively deal with dioxin pollution.

I strongly believe that we need to get to the bottom of this issue not by newspaper leaks, not by charges and counter charges in the media, and not by unsubstantiated claims by private groups, but rather through taking the testimony under oath of EPA personnel and others involved in the Michigan dioxin case.

The public must have confidence that their elected officials are providing factual information upon which rational decisions and actions can be made on the nature of dioxin pollution in Michigan.

Again, Mr. Chairman, I appreciate your convening this hearing today. This is an important matter for my constituents, all residents of Michigan, and ultimately the people of this country who are concerned about potential toxic chemical pollution.

Mr. LEVITAS. Thank you very much, Mr. Albosta. I appreciate your statement. I understand your particular concern in this matter.

If there are no further statements by members of the committee, I would now like to administer the oath to the witnesses who will be testifying today. I trust that copies of the rules of the committee are available and if anyone seeks counsel, you are certainly able to have it.

I would like to ask all of the witnesses who have not previously been sworn—Mr. Hedeman, I think you have, and Mr. Lucero has—if you would please raise your right hands.

Do you solemnly swear that the testimony you will give before the subcommittee today will be the truth, the whole truth and nothing but the truth, so help you God?

[All witnesses responded in the affirmative.]

Mr. LEVITAS. Thank you very much.

Dr. Hernandez, once again, welcome. If you have an opening statement, you may proceed as you so desire.

TESTIMONY OF JOHN W. HERNANDEZ, JR., ACTING ADMINISTRATOR, U.S. ENVIRONMENTAL PROTECTION AGENCY; ACCOMPANIED BY JOHN E. DANIEL, CHIEF OF STAFF, U.S. ENVIRONMENTAL PROTECTION AGENCY, AND JOSEPH A. CANNON, ASSOCIATE ADMINISTRATOR FOR POLICY AND RESOURCE MANAGEMENT, U.S. ENVIRONMENTAL PROTECTION AGENCY

Dr. HERNANDEZ. I do have an opening statement; copies have been submitted to you. I plan to read the first page or two, give you some highlights on the rest of it, and then finish with a summary statement of my own dealing with my appearance here today.

I appreciate this opportunity to meet with you today to discuss the status of the Superfund Program. As we are well aware, the last few months have seen increasingly public and congressional scrutiny of this Agency's administration of the program.

The public charges and allegations about mismanagement of the program have disturbed the Agency greatly, particularly the line staff directly charged with implementing the Superfund Program.

We intend to be open with you and other Members of the Congress as to our problems and as to our accomplishments. We have nothing to hide and a great deal to be proud of. Yet, implementation of the Superfund Program as it was designated by Congress has proven to be more complex than originally understood.

A full and fair assessment of EPA's administration of the Superfund Program must evaluate our current status and problems in the context of past milestones and decisions. We have some very serious issues facing the program, but we have already moved to resolve some of these serious issues.

Certainly, one of the critical elements in the Comprehensive Environmental Response, Compensation and Liability Act of 1980, or Superfund, as it is commonly called, is the close relationship between Federal and State Governments in response actions at hazardous waste sites.

Congress designed an active role for the States as well as for the Federal Government in response at these sites. While it is true that the Federal Government has a large trust fund at its disposal, Congress nevertheless also required States to contribute financially as well as in other ways to these actions.

States are required to give three assurances in order for a remedial project to move ahead. Mr. Chairman, I believe that those three assurances that appear in the middle of page 2 represent the fundamental, critical path elements of future Superfund projects. Let me tell you what they are: First, the assurance of 10-percent share of the costs for remedy at a privately owned hazardous waste site; if it is publicly owned, it is a 50-percent share.

Second, if required for site remedy, the State must provide an adequate off-site, permanent hazardous waste facility for disposal of site wastes. Finally, the State must assure the continuing operation and maintenance associated with any response at a site. This may be decades.

I believe those are the three critical-path elements that the program will come down in time.

Let me quickly move to page 4 and just ask you to look at a couple of numbers. At the top of page 4, it describes a number of our sites where ground water problems are considered to be of great concern--three-quarters of all of the 419.

Down at the bottom of that paragraph it says, because of the ground water problems, we believe that it will take as long as 44 months, or over 3½ years, to address a site where subsurface problems are involved.

Mr. Chairman, one of the things that I gave to some of the members of the subcommittee today was a piece of paper with some words on it, and I find it much easier to follow the Superfund Program with that paper. Let me just give you my version of these numbers. Unfortunately, they do not always gel, by one or two, with somebody else's numbers because we have different pieces of data to work from.

The first thing we have done is to designate 419 sites on the national priority list. We have taken a lot of emergency actions; these have been taken at 48 sites. The end of emergency actions is that the emergency situation is contained, and I think that the containment of the immediate problem is one that we have sought.

The next step can be a removal process where we remove surface contamination, drums and things like that that appear to be a hazard, than to fence the property and things like that. There are 99 sites at which we have had some kind of removal action.

If we move down and ask "at how many sites have we done a total surface cleanup—that is, we have worked on either emergency or removal problems to arrive at where the surface of the ground is protected?" That is 62 sites.

Finally, the remedial—and this is the end product where, by and large, all of that 419 will go through before we get to final cleanup—that remedial business has about six steps that you have to go through, and I want to just quickly list them: a primary assessment; there have to be onsite inspections.

We have to have onsite field investigations; we have to have feasibility studies. Right now, we have 62 ongoing feasibility studies. The next one is initial remedial work. If there is something, where it makes sense to go out and do it right away, at the end of the feasibility study, we would do it.

End of design is the next step, and finally remedial work, and after that O&M, operation and maintenance; and finally, years later, we will come down to a completed site cleanup.

It is a long process with many places in between. Let me ask you to look at just two other places; I will not talk about them. On page 9 of my testimony, I talk about our enforcement actions; I think that is worth looking at.

This morning, Mr. Chairman, I have cited what I believe are the positive aspects and accomplishments of the program, but even these can be subject to some criticism. However, there are many areas in the OSWER Program that are not as rosy and not as productive.

We do have problems, and I think this hearing will bring them out and we certainly will be willing to discuss them with you.

Finally, a note of personal comment to this committee. I come to this job in a very difficult circumstance, and I am keenly aware of the questions that have been raised regarding the Agency's management and objectivity.

You should know, through you, the American people—that I represent the Environmental Protection Agency, and as long as I am the Acting Administrator, I will continue to be committed to getting our staff back to work, including the Acting Administrator; to maintaining the confidence of the American people in EPA as an institution that has the responsibility of protecting their health, our wildlife, and our natural resources; to vigorously implementing our environmental statutes; and to enforcing these environmental laws with a view to speedy compliance.

Let there be no mistake about what I am saying with regard to Superfund or our air programs or our water programs; the Environmental Protection Agency will take whatever action is necessary, however stringent, and use every tool at our disposal to accomplish the goals of the statutes that the Congress has given us.

Thank you, Mr. Chairman. We are open for any questions you may have.

[Mr. Hernandez' prepared statement follows.]

STATEMENT OF
JOHN W. HERNANDEZ, JR.
ACTING ADMINISTRATOR
UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
BEFORE THE
SUBCOMMITTEE ON INVESTIGATIONS AND OVERSIGHT
COMMITTEE ON PUBLIC WORKS AND TRANSPORTATION
UNITED STATES HOUSE OF REPRESENTATIVES

MARCH 16, 1983

Mr. Chairman and members of the committee, I very much appreciate this opportunity to meet with you today to discuss the status of the Superfund program. As we are all well aware, the last few months have seen increasing public and Congressional scrutiny of this Agency's administration of the program. The public charges and allegations about mismanagement of the program have disturbed the Agency greatly, particularly the line staff directly charged with implementing the Superfund program. We intend to be open with you and other members of the Congress as to our problems and to our accomplishments. We have nothing to hide and a great deal to be proud of. Yet implementation of the Superfund program as it was designed by Congress has proven to be more complex than originally understood. A full and fair assessment of EPA's administration of the Superfund program must evaluate our current status and problems in the context of past milestones and decisions. We have some serious issues facing the program -- but we have also already solved other serious issues.

Certainly, one of the critical elements in the Comprehensive Environmental Response, Compensation and Liability Act of 1980, or Superfund, as it is commonly named, is the close relationship between the Federal and State Governments in response actions at abandoned hazardous waste sites.

Congress designed an active role for the States as well as the Federal Government in response at these sites. While it is true that the Federal Government has a large trust fund at its disposal, Congress nevertheless also required States to contribute financially as well as in other ways to these actions. States are required to give three assurances in order for a remedial project to move ahead:

1. Assurance of 10 percent share of the costs for remedy at a privately-owned hazardous waste site; if that site is publicly owned, that State share may exceed 50 percent of the total cost of response.

2. If required for site remedy, the State must provide an adequate, off-site permitted hazardous waste facility for disposal of site wastes.

3. Finally, the State must assure the continued operation and maintenance associated with any response action at a site.

While I think everyone agrees on the need for State involvement from the outset of a project, I do not believe that members of Congress, as well as other members of the environmental community, envisioned the difficulty that States would have in meeting these requirements; nor did we foresee the impact and delays that these would cause for the cleanup efforts at Superfund sites.

Let me give you a few examples. The first assurance required from a State, that of the cost-share, is known to the States at the time that sites are nominated and scored for the National Priorities List. Only

22 States have a continuing established fund to cover such remedial actions. In the remaining States, independent legislative authorization is needed for the required State match at each site. Obviously, this can be a lengthy process and one that may not be concluded by the time the money is needed. In fact this is probably the single most constraining factor in moving the Superfund program at this time.

The second assurance, that of an adequate off-site disposal facility, is also one in which we are running into unforeseen problems. For example, any cost effective remedy for the dioxin sites in Missouri that requires the excavation and disposal of contaminated soil must await the identification by the State of a permitted hazardous waste disposal facility. At present there is no facility that can accommodate large amounts of soil with high levels of dioxin contamination. Certainly, one of the things we are most vigorously pursuing with the State of Missouri, in addition to our efforts at relocation, is the need for a disposal site.

The third problem unanticipated by the Congress and the Agency is the complexity of the physical and environmental problems at these sites, and the uniqueness of such problems. As the Agency began to investigate each of these sites more closely, it became apparent that at many sites ground water is most probably affected by the contamination. Indeed, in almost 400 of the 650 sites that were scored for the National Priorities List, there were observed ground water releases; 328 of the proposed 419 sites would be placed on the National Priorities List based on their ground water scores alone. Naturally, the seriousness of the ground water contamination is increased when the ground water is used for public water supply. The problems encountered by the Agency in investigating and remedying these sites revolve around the fact that ground water is an unseen resource that cannot easily be traced, much less diagnosed. We find that it is necessary to monitor and sample ground water over a number of seasons, possibly for as long as a year, before we have complete information about the extent of contamination and the direction of its movement. Considering that the state-of-the-art technology to treat ground water is extremely expensive and cumbersome, it behooves the Agency to take the time necessary in the study stage to adequately understand the problem and the potential effects of any remedial action before committing Superfund money. But for this reason, we anticipate that it can take as long as 44 months or over 3 1/2 years to address a site when subsurface problems are involved.

This lack of understanding on the part of the Agency and the public as to the speed with which these projects could move has contributed to high expectations and to charges of delay and mismanagement of the fund. The Agency has cleaned up completely five sites, as we have reported. But we intend and are moving ahead on surface actions at sites that will remove the original source of contamination and reduce the direct threat to public health and the environment. We have completed three surface cleanups so far; we anticipate we will have nine more completed by the end of this fiscal year. It is important to note that costs for response to contaminated ground water problems generally will be more than for other types of Superfund remedial actions. Contamination at these sites can extend over many square miles, increasing the scope of the cleanup projects; treatment alternatives can include state-of-the-art technologies or unique applications; and solutions may require capital intensive construction, such as interceptor wells and advanced treatment processes, as well as operation and maintenance for extended periods. Some sites could require decades to maintain the remedial action; this ties back into the third assurance for future maintenance required of States, and the Agency's concern about lack of State funding for Superfund projects. Will States have the ability to maintain and operate these systems once they are installed by the Superfund action?

Another area in which the program is experiencing unforeseen delays is in the area of project start up. There are two ways of managing the projects: either the Federal government or the State government takes the lead role. If it is a Federal lead project, the Army Corps of Engineers will generally manage the design and construction stages, contracting out all actual work to the private sector. If it is a State lead project (and over 60 percent of our projects are State lead), the States will take the lead in implementing the planning or design and construction stages of the project. In fiscal year 1982, 30 cooperative agreements or amendments with the States were executed by the Agency, totalling slightly over $34 million. We are happy to see the States take an ever-increasing role in implementing the

Superfund program effort, but the lack of readiness or proper authorities on the part of States in following through on the necessary procurement actions have lead to unnecessarily long time delays in getting projects started up. For example, the Agency awarded almost $500,000 to one State for a remedial investigation and feasibility study in May 1982. It took the particular State until January 1983 (over six months), to hire the necessary staff to develop a contract proposal, send the procurement action out for bids, evaluate the proposals and award the contract. Similarly, the Agency awarded over $300,000 to another State for an investigation and study and initial surface work in July 1982. The property owner refused to allow private contractors access to the site and the State and EPA are now in court trying to obtain access. Now over 8 months later, the State has been unable to get that project started.

How can these delays be avoided or minimized? States can begin their procurement process prior to the actual execution of a cooperative agreement during the period of negotiation when the general scope of work and conditions have been agreed on by all parties. Illinois, for example, has pursued this course of action. Also, States with many sites, such as New York, New Jersey and Michigan, may combine a number of sites in one procurement action, or use "level of effort" contracts that will reduce the number of individual procurement actions required. Finally, EPA has begun to work with the National Governors Association to sponsor a symposium of State officials to exchange views and share experiences with Federal Government in expediting the procurement process. This will also provide an additional opportunity for EPA to explain the procedures and review the policy for developing cooperative agreements so that process can be expedited also.

Let me turn now to the accomplishments and solid record of progress that the Agency has compiled in the Superfund program to date. The full implementation of this major piece of environmental legislation is a challenging task for the Administration, requiring a working relationship between the Federal Government and the States for response actions; extensive consultation and cooperation among Federal agencies; and a system of accountability to the American taxpayer for the efficient and effective management of Fund monies that finance removal and remedial actions. In the first two years of this program, the Agency had concentrated its efforts on building the necessary management systems, developing a strong policy and regulatory framework, and initiating the field planning activities required for responsible decisions on site remedies. Milestones during this time include establishment of a new Superfund program office within two months of passage of the statute; design of automated data systems for a site inventory and for project tracking; development of a comprehensive financial management and accounting system that will support cost recovery efforts as well as enhance program management; design of a hazard ranking system to prioritize site response; award of major contracts to support the field activities of the emergency, remedial and enforcement aspects of the program; development of an Interim Priorities List to direct funds to site activities to begin swiftly; revision of the National Contingency Plan to reflect the expanded authorities of the Federal Government in the area of hazardous waste response; proposal of the National Priorities List of 419 sites; and health and safety training programs for regional and State employees involved in actual response actions.

We have also continued to implement the emergency or removal authorities given to us in the Superfund act. This aspect of Superfund has not received a great deal of national attention but it is the area in which we can achieve the most immediate results. It is important to note that in removals the Federal Government can act alone. Thus far, we have approved a total of 115 removal actions for implementation by EPA or the Coast Guard; of these, 99 have been completed and over $25 million has been obligated to date. Total surface cleanup has been completed at 62 sites - these are sites in addition to those on the priority list for remedial action. For 48 sites on the priority list, we have had to step in on an emergency basis. In short, EPA has not hesitated to exercise its authorities to protect public health and the environment.

For long term or remedial action, the Agency is similarly compiling a growing record of work underway. As of the end of February, we have obligated in excess of $70 million for remedial actions; these include 62 feasibility studies, 18 designs, 14 remedial actions and 15 initial remedial measures. We've entered into 42 cooperative agreements and 36 contracts.

Under CERCLA the Agency has vigorously pursued enforcement actions to abate the problems associated with the actual or threatened release of hazardous substances and their subsequent cleanup.

Section 106 authorizes the use of administrative orders or civil injunctive relief for the actual or threatened release of hazardous substances. The goal of the Agency's enforcement activities has been to obtain through the use of administrative orders and civil actions as many removal and/or remedial actions as possible without the obligation of Fund money.

During FY 1982, 121 Administrative Orders were issued pursuant to section 106 at six sites. In addition, the Agency negotiated 20 settlements of private party cleanups, valued at approximately $29.3 million. Already in FY 1983, the Agency has reached five settlements involving private party cleanups which are valued at approximately $57.7 million.

Under section 107, owners or operators of disposal sites, generators of hazardous materials and transporters of hazardous materials may be held liable for removal or remedial costs or natural resource damage claims from hazardous waste releases. Private party contribution cost recovery settlements have been reached at 6 sites, with a total contribution of $3.7 million. Through EPA negotiated settlements, States have been able to recover approximately $13.9 million of their own funds.

The Agency also plans to begin use of its authority under section 107 through the use of civil actions, to seek up to treble damages against such persons when there is a failure without sufficient cause to properly provide removal or remedial action upon order by the President. The threat of triple damages provides a strong leverage to the Agency in negotiating site cleanups.

The Agency is currently concentrating its cost recovery actions on those immediate removals which have occurred during the last two years. During FY'83 the Agency plans to inititate 18 new cost recovery actions. Six of these cases have already been referred to the Department of Justice. These 18 cases represent 96% of the Fund money which has actually been spent for cleaning up sites where only immediate removal actions were necessary.

During FY 1982 and the first quarter of FY 1983 the Agency has referred 18 new CERCLA cases under both sections 106 and 107 to the Department of Justice. Twenty-six additional complaints already filed have been amended and referred to the Department of Justice to include §106 imminent hazard or §107 cost recovery actions.

This spring, the Agency expects to issue guidance regarding the pursuit of cost recovery actions. This guidance will include discussions on the assembly and maintenance of evidence, appropriate documentation procedures for processing and negotiating claims, and the mechanics of repayment to the Fund. This guidance will be used by all EPA offices and contractors involved with cost recovery actions in order to maintain a consistent and thorough approach in these cases. It is the intention of the Agency to obtain a complete response in each cost recovery action.

To date, Agency enforcement actions have resulted in a total savings to the Fund of approximately $146.4 million.

And it is no secret that the Agency anticipates an increasing level of effort in the Superfund program. The 1984 budget request for Superfund is an increase of 48 percent over 1983 levels, and an increase in resources for site cleanup of nearly 55 percent. We have always anticipated that expenditures would peak towards the end of the statutory time period for the Superfund program. A simple explanation for this is the Agency anticipated moving into the design and construction phases of substantially more projects at ever increasing costs in the years 1985, 1986 and 1987. The first few years of the program, as we have repeatedly said, have been devoted to building the necessary policy and regulatory framework on which to base our actions and to getting underway the necessary feasibility studies required to diagnose the problem at each of the sites. We propose to start more design and construction projects, the high cost phases of a project, over the next few years. Critics of the program who have accused the Agency of holding down costs and not expending the money of the program do so in a short-sighted manner, forgetting the need to manage the fund effectively so that the dollars will be available for the later, high-cost phases of construction. We believe that we have managed and administered the Superfund program in such a responsible manner.

I wish to thank the committee members for this opportunity to appear before you to outline some of the major problems still facing us in implementing the Superfund program. I would be more than happy to take any questions from the committee at this time. Thank you.

Mr. LEVITAS. Thank you, Dr. Hernandez, for your statement and, without objection, your entire statement will be made part of the record at the conclusion of your opening testimony.

As I have indicated, and other members have indicated, there are a number of areas that will be discussed today, and I am going to start off my questions not on what I call one of the jackrabbits, albeit an important one—we will get around to that in a little while—but on some more general questions about the Superfund Program itself, the role of the Agency, and your role in that regard.

Before I do that, I want to make one statement. Yesterday, I received through my staff a copy of a document which had been submitted concerning the handling of the documents which we have requested, the subpoenaed documents and the others relating to them, setting forth a procedure or means for dealing with those documents which you had signed.

I have not had a chance to examine that completely to ascertain what its effect is, but I think I should tell you that I am at the present time operating under the memorandum of agreement which was signed with Mr. Schmultz, the Deputy Attorney General; and Mr. Fieldinq, counsel to the President.

We would have to make certain that anything we do is not inconsistent with that, or their agreements are modified to reflect what you have proposed. I might say that I have written a letter to Mr. Schmultz and Mr. Fielding.

Dr. HERNANDEZ. Mr. Chairman, could I respond just to that?

Mr. LEVITAS. Let me just finish what I was saying.

Dr. HERNANDEZ. Yes, sir.

Mr. LEVITAS. I have written to Mr. Fielding and Mr. Schmultz, suggesting that the agreements reflect essentially what you have proposed. That letter was received by them last Friday. I have not yet had a response; I hope I will very shortly.

Dr. HERNANDEZ. We have written very similar agreements for the other House committees who have asked for access to these same or other documents from EPA. It was our effort to try to put this into one that was equal for everybody and to restate it.

This agreement that we have proposed has gone through, I believe, the Department of Justice and the White House. So, I hope that they are the same. Our intention was to give everybody access to any EPA papers that they wanted.

Mr. LEVITAS. Well, the document, just based on my initial perusal of it, is very similar to what I had outlined in my letter to Schmultz and Fielding. If they can just get around to dealing with it, I am sure we might be able to move forward expeditiously.

Let me get into my questions. Let me start with a general question. You are obviously no stranger to environmental issues, given your past experience, and you have been involved for the past 2 years with the Agency's Clean Water Act Program, which includes several provisions dealing with toxic chemicals, as well as the problems that are inherent in administering a multibillion dollar effort, as is the Construction Grants Program, which makes up about 60 percent of the Agency's budget and expenditures.

You have also been instrumental in the scientific peer review area, with a high degree of involvement, if I understand correctly, with the Agency's Science Advisory Review Board.

My question is this: Given your activity in the Agency and given all that has gone on not just in the past few weeks or months, but since you have joined the Agency, how do you see yourself proceeding at this point in time? What changes or adjustments do you plan to make that you believe are necessary, and what changes do you plan to make in the Superfund Program and its administration?

It might also be appropriate for you to comment on those problems faced at OSWER to which you referred in your opening statement.

Dr. **Hernandez**. Yes, sir; I would be pleased to do that. I think, as you have indicated, there are a number of areas within the Agency that I have not had what I would call hands-on implementation, simply because I have been assigned other responsibilities to take care of, the Clean Water Act being one of the principal ones.

I think that one of my first goals is to become much more familiar with the operation of the Solid Waste Program and, more specifically, coming from a technical background, I need to learn much more about the statutes and their implementation, the requirements, and a lot more about enforcement.

I have had very, very little experience in that area and just do not know the procedures that people go through. So, in that personal sense, that is the kind of process I will have to go through.

In terms of some of the problems in Superfund that we have noted, there have been concerns about inadequate staffing and inadequate budgets for the regional offices and for headquarters.

The overall money, in terms of funds available for cleanup, there is an ample amount of money there. The funds that we have allocated to do the management of the process; right now, it runs about 15 percent of the expenditures for administration.

I believe the law has some kind of a cap on the order of 21.9 or 22 percent, or something like that. So, we are below that.

I think the kinds of things that I would hope that we are able to do is to break some of these barriers; to go out and work with the States to help encourage them to provide two things: one, moneys to match the Federal funds on a 90-10 basis, and second, site designation.

When we look at the Missouri issues, where in the world are we going to put that stuff is a mystery to me. It is going to be one of the things that will slow the cleanup of the dioxin in the Missouri area considerably until a site is found.

I think that there are a lot of management problems that we have noted. One of the things that I have learned in the last few days is that the kinds of program monitoring that Mr. Cannon's organization does and that Mr. Crampton does for him—they have had problems trying to identify output from the Superfund Program.

That does not mean sites cleaned up; that means programmatic milestones and productivity that you have in terms of accomplishments of individual goals down the line that we look for as environmental markers.

As a part of that, they are reviewing an overall management of the Solid Waste Program, both RCRA enforcement and the Superfund. That is scheduled to be completed sometime in May or April, I guess. I think that will be very helpful in terms of what we need to do in the Superfund Program.

I think some of the other kinds of things that people have identified. Right now, we have a centralized management on Superfund. I do not think that was unrealistic for the 1 year or 18 months of the program when we were, trying to get a handle on where we were going and what we were doing. It was vitally important.

I believe that this may be a time to decentralize to some extent. These policies will have to come through the kind of debated issues of how we would do it, what kind of differing relationships we will have, and what kinds of communications. But I think that stuff is in place and minor modifications can accomplish that.

Paperwork is always a problem, and when you send everything back to a centralized headquarters, it becomes difficult to use.

I think that in the personnel area, while we may have an adequate number of bodies, we may not have the right kinds of skill mixes. We need to relook at that and provide some of the best people that we can in the OSWER Program.

I could identify others, and I am sure we will go through with them. That is a kind of a summary of the things——

Mr. LEVITAS. Well, let me ask you about two points that you touched on. One that surprises me, really, is you just stated that there are ample funds for cleanup. Now, we understand there are about 14,000 to 20,000 sites, but let us use 14,000. There are 419 on the priority list, which was increased from 160.

It is my understanding, and Mr. Cannon may be able to be of some assistance to us on this, that based upon your current analysis, the Superfund, as it is presently constituted, would not result in the cleanup of more than 170 sites.

Dr. HERNANDEZ. I was thinking a much narrower timeframe. Living to the end of this fiscal year will be a triumph, and I was thinking about fiscal year 1983. We have more money for the cleanup of sites than we can probably allocate in this fiscal year.

The long-term part of the program is another issue, and one that we are addressing and one we have had some discussions already on the policy issues. We have made some estimates on how much would be in the fund totally—the $1.6 billion that comes out the revenue source and the Treasury, interest that we will get off of having some of that money available in banks, and then the last one would be recovered funds,

The guesses that I have seen are that by 1988 we will have something like $1,900 million totally available to Superfund.

Mr. LEVITAS. Well, I do not want to get away from this because I think it is extremely important. The American people are very concerned about the cleanup of these hazardous waste sites, and they are becoming more concerned as they learn more about it.

Is the Superfund going to have enough money to cleanup the hazardous waste sites that have been, much less will be, identified?

Dr. HERNANDEZ. We are talking about at least the 419 or so.

Mr. LEVITAS. For openers.

Dr. HERNANDEZ. For openers. I think that the estimates that we have are that the $1.9 billion that we will have will probably cover less than 100, not 170, this is our current estimates.

Part of that is, and you can pick up the newspaper and find it, $33 million to Times Beach alone, and that certainly throws your estimates of how much per site off the track. PCB's in the Hudson may eat up another large piece.

I think that as I recall the law on this piece, and you can help me, I believe we have to have a report to the Congress at the end of the fiscal year to give you some kind of an estimate as to how we view the work that can be accomplished with the moneys that will be available.

I think by that time, we will be able to give you some kind of a realistic estimate for future needs, and perhaps some options. I think one of the things we need to do, and we cannot do this by 1983 or 1984, but someplace before the moneys run out, through our assessment, give the American people some kind of an idea of what kinds of things remain out there that need to be done that pose significant health hazards.

Mr. LEVITAS. But, specifically, can you or Mr. Cannon or Mr. Hedeman tell this subcommittee, will the program as it is presently constituted and presently funded, cleanup the 419 priority list sites?

Dr. HERNANDEZ. I think the answer to that is no.

Mr. LEVITAS. The answer is no?

Dr. HERNANDEZ. No; more like 100.

Mr. LEVITAS. More like 100?

Dr. HERNANDEZ. Yes.

Mr. LEVITAS. And we are looking at thousands of these sites, and I think that is what this committee is really targeting on. How can we assure the American people that their waters will not be poisoned because the program is not adequately funded or structured?

Let me turn to another problem. During recent hearings——

Mr. SNYDER. Mr. Chairman, before you leave that, let me just ask a question?

Mr. LEVITAS. Sure. Mr. Snyder.

Mr. SNYDER. Will not the adequacy of the funds depend upon the effectiveness of the enforcement recoveries? We do not really know what that is going to be but, theoretically, if you could find every polluter and they were all solvent and you could place liability, you would recover all the cleanup costs.

So, that is a factor that in this exchange of questions has not been looked at.

Mr. LEVITAS. I think that is correct, but it is also my understanding, again, based on testimony which Mr. Lucero and Mr. Hedeman previously gave, that they are looking at approximately an equal number of sites where there will be recoveries to finance the cleanup.

It is about 50-50-50 percent Superfund, 50 percent recovery funds—and that comes up, using Dr. Hernandez, most recent figure, to about 200 out of 419 priority sites. We are still a long way from achieving what I think the American people are looking for.

These are the people that I think are best capable of telling us what the problems are so we in Congress can make some decisions on where we go from here.

Mr. SNYDER. Well, that is right, and if there is an inadequacy in the fund, that is our responsibility.

Mr. LEVITAS. That is right.

Mr. SNYDER. We gave them until December 1984 to come up with the figures as to where we ought to be going with superfund.

Dr. HERNANDEZ. That is correct.

Mr. SNYDER. I thank you for yielding.

Mr. LEVITAS. Thank you. I am advised that the static noise may be coming from the photographers; we will try it one more time.

One other problem that was called to our attention rather forcefully was that there are problems such as conflicts that existed between Mr. Perry's office, the Office of Legal Counsel and Enforcement, and Ms. Lavelle's office at OSWER; that two officials of equal standing in the Agency's hierarchy had impasses between them as to what approaches should take precedence—negotiate first and litigate second, or litigate first and negotiate later, or using the Superfund to go and clean up and then go after people and have them contribute.

My question is what are you going to do about this problem?

Dr. HERNANDEZ. Well, I have a very easy management style. If people do not work together and get along and solve problems, somebody goes. It is a very easy management style, and I have used that a lot in the past and I do not mind using it in the future.

But I also help facilitate things, and part of that understanding of the policies that we will follow has to come about through that kind of coordination between the enforcement people and the program people, and I hope to be a big part of these discussions so that I understand the issues and can make the right judgments.

Mr. LEVITAS. My time has expired; I will come back to some other questions later.

Mr. Molinari.

Mr. MOLINARI. Dr. Hernandez, as you know we have had problems in trying to secure EPA documents.

I am hoping that maybe you can shed some light on some of these areas for us. I ask you because there was a meeting on October 1 of last year that was attended by yourself, the chairman, Mr. Levitas, Tom Hagedorn, who was then ranking minority member of this subcommittee, and myself.

This occurred after we were stopped in our search for various documents. We had a very cordial meeting that lasted maybe an hour or two, and at the conclusion of the meeting, you stated that you would see to it that we would get any and all documents.

About 4 or 5 days later, we were stunned and surprised that, again, there was a reversal in the decision to allow us access to the documents. Suddenly, we became aware of terms like "enforcement-sensitive," "redacting" and others.

What happened after that meeting that prevented us from gaining access to the documents?

Dr. HERNANDEZ. I believe that part of that record is already available, but let me tell you about my own understanding of the issues. I think there were some intervening things that took place;

people went to New York, and so on. But the most important meeting was one that took place at EPA, where Mr. Prolman and another one of the staff visited Mr. Brown, who is sitting next to me here, and with Mr. Ramsey from the Department of Justice.

Mr. Brown has written up a summary of that, and I think one paragraph will tell you why things had changed. Mr. Brown reports, "The meeting took place in my office. I explained to the subcommittee staff that our previous informal arrangement"—that is the one that we had negotiated with them—" had been changed by virtue of a subpoena that EPA had received from the Oversight and Investigations Subcommittee of the Energy and Commerce Committee of the House of Representatives.

After receipt of a formal demand for copies of documents EPA and the Justice Department deemed to be enforcement-sensitive, it was decided that the position of the Administration would be to deny access to or copying of such materials. I stated that the position had to be uniform throughout the Administration for all committees of the Congress.

I think it was probably clear at that point that this was not an EPA issue where I could be involved and negotiate, but it had been moved to the status of being an administration policy.

Mr. **Molinari**. Doctor, let me interrupt you, if I may, because I have limited time. I am talking about October 1 when we had the meeting which resulted in an agreement. I thought after that, we were going to get all documents.

Did you go back following that meeting and make a report to the Administrator as to what transpired?

Dr. **Hernandez**. No, not in great detail, but I presume that Mr. Daniel, who was also at that session, and Mr. Perry, who was at that session, did.

Mr. **Molinari**. What session are you talking about?

Dr. **Hernandez**. October 1. They were at the October 1 meeting with us, and my understanding is that things were terminated. And then the explanation for that came at this October 29 meeting when——

Mr. **Molinari**. I am advised by counsel that the formal rejection was on the 29th of October. Now, this was well before the subpoena was served.

Dr. **Hernandez**. Yes.

Mr. **Molinari**. What I am trying to determine is who was responsible for that change. Was there a memorandum prepared concerning the meeting we had that day?

Dr. **Hernandez**. Yes, I believe that is correct. I have seen a letter from Mr. Perry to the chairman outlining what the agreement was. By and large, it sounded like what we said, more or less. So, there was a letter that we knew about.

Mr. **Molinari**. Was any memorandum written by anybody at that meeting from EPA and addressed to the Administrator or anybody else, other than our subcommittee?

Dr. **Hernandez**. Not that I know of, but I am sure there may be some notes.

Mr. **Molinari**. Is there anybody here today—Mr. Daniel, I notice, is here—that would be aware of that?

Mr. **Daniel**. Mr. Molinari, there was a verbal report given to the Administrator. There was no written report of that meeting.

Mr. **Molinari**. I cannot hear you, Mr. Daniel.

Mr. **Daniel**. There was no written report given to the Administrator. A verbal report was given of all of the discussions we had that day, and they were given to her completely.

Mr. **Molinari**. I would like to just quickly turn to the question of policy and your statement that we may not have the right kinds of skills. I am pleased to hear you say that if changes have to be made, you are going to make them.

Last week Mr. Perry was confronted with a memorandum that was issued August 13, 1982 directed to the 10 regional offices telling them point blank that if they did not send more cases for referral for litigation to Washington, not that they were going to be replaced or fired, but that they were going to have their staff resources cut back.

Region II, has some of the more serious problems in the country. New Jersey is part of that region and ranks No. 1 in the number of priority sites. I believe it has 69 out of 419 sites. They were going to cut the full-time employees down from 27 to 12 if the quota was not met.

Nobody gave any thought to the fact that maybe the regional administrator or maybe the regional counsel were not doing their job. Maybe the reallocation or redistribution of resources should go in accordance with where the serious problems are.

Instead, it was going to be based on what he called performance or lack of performance. Now, it seemed to me that that was a terrible policy decision. If you did not have a good regional administrator and a good regional counsel pursuing what they were supposed to do, you are going to prejudice the citizens of that region by cutting back on personnel.

I would like to have your response to that so-called policy decision, and Mr. Cannon's as well.

Dr. **Hernandez**. Well, I think one of the things we try to do is to judge people's performance by some kinds of markers, some kind of productivity. When you are dealing with 10,000 people, you have to have some kind of goals that people achieve things by.

I think that Mr. Perry was trying hard to do that.

Mr. **Molinari**. I do not question how hard he was trying. I do question how much sense this policy makes. It disturbed me deeply and, frankly, it did not make any sense whatsoever. It was absolute insanity to turn around and suggest that you were going to base your redistribution of resources on case referrals. What you are doing is you are blackmailing them into sending cases to you for prosecution.

Dr. **Hernandez**. I think one of the rationales sounds like this; it says, "How many cases are you working on?" They say six. "How many enforcement lawyers have you got?" "Sixteen." You know, that is three per person. It says, "Come on, fellows, let us be a little more productive."

Mr. **Molinari**. Well, it was a little stronger than that, Dr. Hernandez. I even had a chart showing a thermometer and the goals. In region II of all places, where they said "we need your help," they were worried about being cut back and being able to perform the job.

So, it was not a question of saying, "Come on, fellows." What he was saying was, "Your jobs are on the line and you are going to have your office cut into less than half of those that you have today."

Now, cannot we agree that some of these dump sites may be very complex in the treatment that you have to give to them, so that indeed you may have to devote a great deal of resources and attention to on a particular site?

When you play this numbers game, it really does not come out right because you may have a number of sites where they can quickly submit them for litigation because of the legal background. But in other sites, it may take the resources of almost the entire region to deal with one site.

Now, that bothers me deeply and I would like to maybe turn to Mr. Cannon because I am not satisfied with the answers that I have been getting.

Mr. CANNON. Well, I am not familiar with the memorandum at issue; I do not remember reading it. And I did not hear Mr. Perry's testimony, but to the extent my office got involved in this issue, I think we had a discussion over what we call internally a workload modeling.

And there was originally, as I understand it, a proposal to develop the workload analysis of the regional counsel's office based on this variable of cases turned in. But my own recollection of it is a new workload model had been developed between people on my staff and people on Mr. Perry's staff that took into account more than this single variable.

And that is the extent of my knowledge.

Mr. MOLINARI. Wouldn't commonsense say, let us take a look at the performance of these people in the region. If they are not doing the job, let's fire them, instead of prejudicing the rights of all the citizens in that region no matter how great a health peril exists.

There was no mention whatsoever in the memorandum about the quality of performance, and perhaps they did not have the skills, which is what Dr. Hernandez said before, and that if the skills were lacking, it would made sense to get somebody else in there who could do the job.

There was no reference made to that at all. It was strictly a numbers game based on the number of referrals you send to headquarters for litigation, and that is going to determine the regions resources in fiscal year 1983.

Mr. CANNON. Well, as I say, I have not—I am not—I have not read that memorandum, but as I say——

Mr. MOLINARI. Dr. Hernandez, let me throw the ball back to you. You are Acting Administrator.

Dr. HERNANDEZ. I think the——

Mr. MOLINARI. You are Acting Administrator. We have a serious problem here. Certainly the area that I represent has problems in many places. I am deeply concerned when I read a memorandum like that suggesting that we are going to go from 27 full-time employees down to 12. There is no way they can do the job.

What do you do about evaluating performance at the regional level?

Dr. **Hernandez**. By and large, that is done through two techniques. The regional—the general counsel has cognizance over all of those lawyers out in the regions, not the regional administator. And that is why part of that that you see flowed in that direction.

I really believe—I have heard you. I understand your concern. Take a look at it. You know, my own management style is different, and I would hope that we can provide that kind of mixed forms of evaluation. And I think one of the problems that we had in the Superfund Program, overall, in terms of evaluating output—I do not mean things cleaned up and that kind of thing, but those other kinds of programmatic goals—has been that they are unique, growing, changing. It is a new program.

We need to find better ways of doing it, and you have brought some to our attention.

Mr. **Molinari**. Well, my time is up for now. It seems to me that somebody has got to give some attention to the quality of the personnel at the various regions. I would like to go into this and I will later on. I know there are some regions where you have had serious problems with the personnel and the activities of those personnel. But nobody in the past has seemed to make any suggestions about changing those personnel.

We go helter-skelter down the road letting them make the same mistakes, and it seems to me that that is an area that we should be talking about—redistribution of resources based upon where the problems are, not on the performance of the individuals who may or may not know how to do the job.

Mr. Chairman, I will pass at this point.

Mr. **Levitas**. Thank you, Mr. Molinari. The gentleman from New Jersey, the chairman of our committee, Mr. Howard.

Mr. **Howard**. Thank you, Mr. Chairman. I am not going to get that deeply involved in here because you have spent more time on this than I have. But it does seem that in EPA's looking over this that the first goal here, and maybe in other places, seems to be not how do we accomplish the job we have to do. Or in this case, how do we clean up the sites?

The first goal seems to be: How do we get the Federal Government out of this completely? How do we relax that. And there are statements that I have here, particularly one in a memorandum from a Kate Bouve, a comment on the draft administors guidance. It says for 1984 and 1985. It notes, "Identify and develop feasible options including realistic Federal options for significantly decreasing active Federal involvement in the program."

Now, is there any program beyond that as to how the job will be done if the Federal Government gets itself out of the activity?

Dr. **Hernandez**. I really do not perceive that. You know, I perceive us doing a lot of things, through enforcement goal out there, that as we begin to do some cleanup, I think that will have to be there in order to wield the tools.

Mr. **Howard**. Well, just based on what Mr. Molinari has said and been leading to, aside from who sent what memorandum and how, but if the activity was going to be that in some way if you are not doing more of this thing we want you to do, you are going to have fewer people with which to do it.

What does that signify the end result is going to be?

Dr. **Hernandez.** I think it was a sincere effort to encourage enforcement activities. I just read it that way.

Mr. **Howard.** But if you really meant what you said, then actually there is going to be less done. The less you do, the less ability you are going to have to do more, and therefore you are going to have fewer people, and you are going to have less done, and the whole program is going to fall apart.

Do you get that kind of a logical conclusion to the thing that Mr. Molinari brought up?

Dr. **Hernandez.** It sounds like a negative incentive program. I think we ought to provide positive incentives wherever we can.

Mr. **Howard.** That is all I wanted to comment on. Thank you, Mr. Chairman.

Mr. **Levitas.** Thank you, Mr. Howard. The gentleman from Kentucky.

Mr. **Snyder.** Thank you, Mr. Chairman.

Dr. Hernandez, I take it that this is your first public appearance since the morning news in the morning newspaper, and I think under those circumstances, it might be appropriate to use this forum to see how you would like to respond to some of the recent events or some of the recent publications and before doing that, though, for the record, would you tell us when you came on board with EPA, the date?

Dr. **Hernandez.** I became the Deputy Administrator of EPA on May 20, 1981. I became the Acting Administrator, I think it was March 8, 1983.

Mr. **Snyder.** But your first employment with EPA was May 20, 1981?

Dr. **Hernandez.** Yes.

Mr. **Snyder.** I wanted to get that in the perspective of the Dow events. Now, it has been alleged in the paper and in the electronic media that you directed that references to Dow Chemical Co. in a draft report on dioxin in the Great Lakes be removed, and that Dow comments on the draft report be incorporated in the final version.

In connection with that, did you at any time direct anybody to remove Dow references or incorporate Dow comments in that report?

Dr. **Hernandez.** In the terms of saying, "you must" do these things, absolutely not.

Mr. **Snyder.** Is it possible at all that you may have done something that might have indicated that to someone?

Dr. **Hernandez.** I would hope that people would not have read, even that I asked them to consider Dow's remarks or comments or, but no more than that. No more than you would consider any other kind of appropriate input.

I think that part of that, if you would let me go on——

Mr. **Snyder.** I would like for you to amplify in any way you want to. This is, I think, your first public opportunity to respond, other than the press release which was issued yesterday.

Dr. **Hernandez**. Much of the information in that report is based on Dow studies. And these are taken out of their reports and then rereported to them.

Let me tell you why I, what I think there are the two decisions I am being criticized for. One of them was to make the report available to Dow. Let me give you my thinking on that particular one. There was a newspaper report. I do not have the exact date, but it is sometime in June.

Mr. **Snyder**. Your press release of yesterday says a June 13, Canadian newspaper report.

Dr. **Hernandez**. Yes. And that is the one I have in my hand, and the title of this is "Ban Fish Containing Dioxin Report on Great Lakes Urges." And this newspaper report has a number of elements in there that raise concerns both within people within the state of Michigan, congressional people from the State of Michigan, and certainly the Dow Chemical Co. The following kinds of statements led Dow Chemical to contact me shortly after it appeared in the press.

And I am reading from this newspaper report. It says, "The report says that Dow Chemical of Midland, MI has extensively contaminated its chemical factory with dioxin, and the chemical related dibenzoferans," and it is in quotes now, "and has been the primary contributor to the contamination of the Tittabawassee and the Saginaw Rivers and to Law Huron." The report is reported to be a draft document that was leaked to the press on the basis that it would not have seen the light of day, and the newspaper article says, "One researcher said the report was leaked; otherwise, it probably would never have seen the light of day."

Shortly after this appeared in the press, I got a number of calls and was concerned for a couple of reasons. One we were dealing——

Mr. **Snyder**. Let me just interrupt briefly. Would you make copies of that newspaper article available for the record?

Dr. **Hernandez**. Yes, sir.

Mr. **Snyder**. Thank you.

Dr. **Hernandez**. We had—some of the offices at EPA had received calls from the Governor's office in Michigan requesting copies of this report. And I cannot remember the exact circumstances under which I was contacted by Dow. My recollection was that I saw one of their people; I believe it was Charlie Sercue in the building that day or the day after that this became known.

And he said, "gee, it really has put us in a bad position. We are being charged with contaminating these two streams, and we do not have a copy of that report. And we certainly would like to see it for us to respond."

The report, having been—much of the details of the report having been in the press and the request by the Governor of Michigan to see it, and a belief on my part that this would be subject to FOIA requirements, compelled me to let them see the report.

Mr. **Snyder**. For the record, FOIA means they could have gotten it under Freedom of Information Act?

Dr. **Hernandez**. That is correct. I believed that. And I think that the staff at EPA believes that a draft report that has gone out in the press like that would be available. There were a lot of other things that were happening at the same time.

When I took a look at the report after I got it, I was irritated by a number of things. First, much of the document contained information about 2,4,5-T, a chemical that is contaminated with dioxin, and there had just been 50,000 pages of testimony on 2,4,5-T and dioxin taken at an administrative law hearing here in Washington, DC.

We had a staff of people who were well informed on that subject. I was not sure that what was in this report coincided with the conclusions that we were to draw, and in the process—we are in the process of drawing-up our conclusions of those 50,000 pages.

It appeared to me that what was in the first part of the report might be at odds with what kinds of conclusions we were going to draw on the basis of that large body of information.

Another of the problems that I saw with this report had to do not only with my concern that statements, like I read, that Dow is responsible for this were not proven in the report, but that the conclusions that should go from much of the report deals with sources. Much of the report deals with fish studies, and things like that and fish contamination. But the conclusions of the report struck me as being not necessarily proven in the report when I read it.

Let me just read you two that lead me to this conclusion.

Mr. SNYDER. Is that the report, Dr. Hernandez?

Dr. HERNANDEZ. This is the original. I think you ought to have a copy of it.

Mr. LEVITAS. Yes. I would like to have a copy of it, and without objection, that report will be made part of the record.

Dr. HERNANDEZ. And I have some letters that are on top of this I would also like to give you because I am going to read from parts——

Mr. LEVITAS. Without objection.

Dr. HERNANDEZ. The conclusions of the report state—and they are talking about dioxins and things like that—it says, "One: Major environmental contamination with PCDDs" and that is a form of dioxin "and PCDFs, particularly with the 2,3,7,8—TCDO isomer." That is the most toxic of all of these so-called dioxins. There are 75 of them and 22 of them that have four chlorine molecules on them . . . Is due to the release of commercial products containing these contaminants; and secondarily, from the combustion of certain chlorophenolic and similar compounds, that is, PCB's.

I am not sure that that was a technically correct statement. I had concerns about that.

> Two: sources like automobiles and coal-fired power plants do not seem to—do not appear to be insignificant sources.
>
> Three: the Dow Chemical Company of Midland, Michigan has extensively contaminated their facility with PCDDs and PCDFs and has been the primary contributor to the contamination of the Tittabawassee and Saginaw Rivers and Lake Huron.
>
> Four: the differential contamination of biological samples from the Great Lakes, being largely confined to Lake Huron and Ontario, suggests that industrial contamination is responsible.
>
> And fifth—

and this is the one that bothered me, I think, probably from a scientific point of view. I said:

The consumption of fish from the Tittabawassee, the Saginaw River, Saginaw Bay, and possibly other sites in the Great Lakes should be prohibited.

Now, to me that was an enormous leap in faith from studies on fish taken basically in these river basins to that they should be prohibited from possibly other sites in the Great Lakes. The Great Lakes is a huge body of water that has great variation of environment. Certainly down around parts of it that are industrialized like the Midland, Michigan area, the Detroit area, the Cleveland area, concerns on those localized places.

But to jump to that kind of conclusion would lead someone to be very concerned about the people that wrote this—and it was out of a regional office. It is not a major EPA report. It was not backed up by contractor works. We did not have 2 or 3 years of things back there. I was concerned about the accuracy of the report and the capability of the people to draw conclusions.

This report had not been through a complete peer review. I would like to read just one or two letters that went through our review process long before I ever saw this report. I should not say long—before I ever saw this report.

On May 29, 1981, a headquarters person named Paul Brown—and he is still at EPA. And he is the cochairman of the Chlorinated Dioxin Work Group—wrote to the region V toxic substances people.

And he says, "The Chlorinated Dioxin Work Group has concluded its review of the draft dioxin paper which Dr. Milton Clark * * *" In most cases members of the group wrote their own comments on copies of the paper.

"I have combined these comments onto one." And that is the one that you see here, that I will give you.

"Two of our members commented in separate memorandum which I have attached. I have one additional suggestion. Separate the scientific"—that is, pages 1 through 27—"from the regulatory policy"—that is pages 27 and 28—"sections of the paper and include the regulatory policy issues as a separate cover memorandum from you describing the paper."

This would help keep policy materials safer from FOIA release and emphasize the responsibility of the agency decision makers who, presumably, are much more reluctant to alter the scientific portions than the regulatory issues portion.

I believe that the people who wrote this believed that it was available under FOIA. I believe that it was. Sending it to Dow was, I believe, the only good faith thing that you could do.

I would like to refer, just briefly, to the second of these written documents. Mr. Chairman, I think that this is important. We have alleged that this is my place to show that I have not done anything that was bad judgment or in some way constituted some kind of violation of law, and I really believe it is important to put this out.

Mr. Levitas. Well, I certainly agree. And I think that is what Mr. Snyder is giving you the opportunity to do. But I do not want you to think the fact that I am, and maybe others are remaining silent means we agree with your characterization. The fact that it was available under FOIA is not the equivalent of making a decision to send it to Dow.

Now, I want you to have this opportunity.

Dr. HERNANDEZ. You have to make those decisions; you have to do them every day on things like this. I believed it was available, and that it was the best piece of judgment.

Mr. ALBOSTA. Will the gentleman from Kentucky yield for a question at this time?

Mr. SNYDER. Yes, sir.

Mr. ALBOSTA. Dr. Hernandez, why was not more scientific people other than Dow's asked for information about this?

Dr. HERNANDEZ. You know, I have said, that is probably the one——

Mr. ALBOSTA. Why, though, why? You must have a reason.

Dr. HERNANDEZ. No; I did not have a reason. I just did not think that that was part of it. You know, today we have a well-described process for obtaining peer review to documents like this.

Mr. ALBOSTA. OK. Would you describe that process?

Dr. HERNANDEZ. Yes. It is a multiple step sort of a thing. The author of a paper like this would take it to his supervisor and say, "I believe that this paper is now far enough along, ready to go, that everything is near ready for a publication."

He would then identify at least two scientists who had nothing to do with the process, nothing to do with making it up, who would—at least two, who would review it. If it is—and they go through and peer review is a process. It does not say, well, I looked through your stuff and it looks OK. It is a very detailed process to make sure that every statement that you have in there is backed up either by a reference that is well cited or by new evidence that is in the text of this thing; that you do not make conclusions without having a good foundation for them or you do not lead to the next step.

Now, that process is well laid out, and that was put into effect in the fall of 1981.

Mr. ALBOSTA. Were there not other people, though, Doctor, that you could have asked this information of, also, to give some comparison?

Dr. HERNANDEZ. Internally, in the Agency it went back to a number of them, people who had been involved in listening to those 50,000 pages of testimony on dioxin related issues on 2,4,5-T. Internally, in the Agency it went to many people.

Mr. SNYDER. If I might reclaim my time——

Mr. ALBOSTA. I thank the gentleman.

Mr. SNYDER. I know that with these long answers I am going over my time, but I often confine my questions solely to a specific subject, so I would like to keep the continuity going with the committee's indulgence.

I think it is significant that you came on board May 20, and you made the decision to accede to Dow's request in mid-June. Your press release of yesterday indicates that in retrospect it might have been better to have circulated it more widely, and I think with your additional experience on the job, that you would have done that today. Now, you also indicated that the Governor asked for a copy. Did you indicate that the Governor or his office ought to have a copy?

Dr. HERNANDEZ. Yes, sir.

Mr. SNYDER. Did anyone else request a copy?

Dr. HERNANDEZ. Not that I know of.

Mr. SNYDER. There has been a report that the University of Michigan had a copy. Do you know about that?

Dr. HERNANDEZ. No; but quite possible.

Mr. SNYDER. Did you refuse to give a copy to anybody?

Dr. HERNANDEZ. I certainly, personally, did not, and I do not think, and I did not instruct anybody not to give anybody a copy.

Mr. SNYDER. Did you ever speak to Don Barnes or Val Adamkus, the region V administrator, with reference to deleting Dow references or incorporating their comments?

Dr. HERNANDEZ. I talked to Mr. Adamkus a couple of times on the issue. I expressed my concern, first, about the report having been released unofficially. It leaked out of the house and that we needed to keep better care of draft reports like that.

Second, that the conclusions, I did not feel were backed up by things, and that if we could not back up the conclusion that Dow was the source, we should not put it in, and that they should consider suggestions made by Dow. Now, I have never seen those suggestions, have no idea what they were, or what process went through to get them into the report.

They did not come to me, and the——

Mr. SNYDER. Then, it is your testimony—you did not make the determination that the Dow comments that were in the draft report were to be deleted or that the comments they submitted were to be incorporated in the final draft?

Dr. HERNANDEZ. Certainly not.

Mr. SNYDER. Is that your testimony?

Dr. HERNANDEZ. Yes, sir.

Mr. SNYDER. Who did that? Do you know?

Dr. HERNANDEZ. Well, I think that as you go through drafts like this, you take the best advice; on this draft copy there are dozens of markings there that suggest changes, and so I think they incorporated these changes.

Mr. SNYDER. Does the draft report in its markings indicate who suggested those changes?

Dr. HERNANDEZ. This was a review by the Chlorinated Dioxins Work Group, here at EPA, done in late May. And so I am sure they took those into account. Further comments that they received from other people; I did talk to Dr. Barnes about some of the concerns I had in terms of both the references to 2, 4, 5-T, because of that large body of testimony that we had had, and also about the conclusions reached, whether or not they could be sustained.

Mr. SNYDER. Did anyone who had knowledge that you were suggesting that Dow get a copy of the report say that ought not to be done?

Dr. HERNANDEZ. No, sir; and now we are 18 months later, and this is the first time that I have heard of these complaints at all. This arose, I believe, after I became named as the Acting Administrator.

Nobody has ever come to me and said, "do you know what Dow is trying to do to us? Do you really want us to put that stuff in?" Nothing like that at all. I had—this issue, I thought it was a very minor kind of thing. If people wanted a copy, gave it to them.

Mr. **Snyder**. Was the procedure that was used in this instance where Dow requested a copy of the draft dissimilar in any way to other procedures that might take place in other cases, or was it similar?

Dr. **Hernandez**. Well, you know, you have to remember to keep this in context; there was no enforcement action here. There was no regulatory action ongoing. This is a scientific paper, or purports to be a scientific paper on an issue. I would hope that scientific issues in papers do have some kind of peer process, both within and without the Agency for their review.

Mr. **Levitas**. If I could, Mr. Snyder, the second bell has rung. Why don't we go and vote and come back, and I will let you complete your question.

And at that time, if you would, Dr. Hernandez, respond to Mr. Snyder's last question, which I believe was: Was this treated differently from other matters, which I do not believe you did respond to.

The subcommittee will stand in recess pending the vote, and we will reconvene immediately.

[Brief recess.]

Mr. **Levitas**. The subcommittee will please come to order.

Before we proceed, Dr. Hernandez, it might be helpful if the court reporter could read back Mr. Snyder's last question so it could be accurately stated.

[Whereupon, the reporter read from the record, as requested.]

Dr. **Hernandez**. Mr. Chairman, Mr. Snyder, at the time that I made the decision to send it to Dow, I certainly did not have a very large body of experience as to what was usual or typical or had been done in the past, so I cannot say much about the past.

Let me say that in today's climate, a similar report, as a draft report that had basically scientific information in it like this, that was not related to any kind of enforcement issue, the reason for not sharing it with someone would be quite unusual, unless it was in such a draft state that it would be prejudicial to its final version. Now that we have a peer review system in place, it is easy to say that a draft of this document did escape, did go out. We are not responsible for that. It is in the peer review process and will come out through that process and would then be available to everyone. We did not have a uniform peer review process at EPA until this administration put it in effect, and so there was no method of getting through to clean up this report.

Mr. **Snyder**. So you are saying it would be handled differently today.

Dr. **Hernandez**. It probably would be handled differently, although there are many, many reports that are handled on a system just like this.

Let me make one note, that if this had been related to a Superfund site by some reason—not an enforcement document, but an information piece—this would have been available to the Levitas committee as part of an ordinary process and available to everyone, and not enforcement-sensitive, and would have been another piece of information out of the files.

Mr. **Snyder**. Dr. Hernandez, we have a copy of the report with some attachments that was handed to us during our recess on voting, and I see a memorandum from a Larry E. Longenecker. Is that how you say his name?

Dr. **Hernandez**. Yes, sir.

Mr. **Snyder**. Who is he?

Dr. **Hernandez**. I do not know him personally at all, sir. I presume that he is in our Toxic Substances Program.

Mr. **Snyder**. It says "Chemical Control Division." Would he be in Washington or in region V?

Dr. **Hernandez**. Yes, sir, he would be here in Washington, DC.

Mr. **Snyder**. Just a quick look at that indicates he has quite a few concerns about it. There is a sweeping statement that "The acceptable daily intake of TCDD should be . . .," such-and-so, and another comment:

> There is in this report an overabundance of self-reference. I do not believe that EPA is well served by quoting itself on all the major questions of dioxin. More primary literature on health effects is needed if this report is to substantiate health hazards.

"Third, the report may cause an over-reaction," and he would have to conclude you should eat no fish caught in waters in the area.

Could you make any scientific observations on those statements?

Dr. **Hernandez**. Mr. Snyder, I think some of the concerns that Mr. Longenecker expresses here on the scientific validity of the conclusions were some that I certainly shared, and I believe that people reading this would come to the same kinds of concerns as Mr. Longenecker had.

Mr. **Snyder**. Would it be a proper assumption on my part—and if you do not know, would you supply the answer—that Mr. Longenecker is a career employee as opposed to a political appointee?

Dr. **Hernandez**. He is a permanent career employee, and I believe he is still with the Agency.

Mr. **Snyder**. I see.

Mr. Chairman, I have gone well over my time. I will catch up the next time around. Thank you.

Mr. **Levitas**. Thank you, Mr. Snyder.

Dr. Rowland.

Dr. **Rowland**. Mr. Chairman, what we are concerned with here, I think, is the hazard that these various chemicals, whether they be organic or inorganic, pose to the health of the people of this country. These are chemicals that leach into our groundwater supply, and over a period of time, may build up.

We do know that there are some chemicals that may manifest their toxicity relatively early, such as thalidomide, a problem that we had a great deal of difficulty with, or they did in England, and then there are those like lead, that take many, many years before they become manifest as toxic agents.

Dr. Hernandez, based on your background and academic training and your professional experience over the past several years, I am sure you can appreciate, possibly more than any former administrator, the technical problems associated with the setting of standards for toxic chemicals, be it for controlling toxic chemical discharges in the surface water, or regulating new chemicals entering the marketplace, or setting drinking water standards.

There are two issue areas that are of interest in this regard. One is the viability or reliability of animal tests for setting those standards for regulating toxic chemicals, and the other is the use of the linear, or the one-hit approach for setting a standard—that is, the assumption that there is no threshold or safe level for highly toxic, carcinogenic compounds.

It is my understanding that for the past several years, the policy in EPA has been to ensure that the public's health is very carefully protected from these most dangerous chemicals, or from chemicals that are suspected of being dangerous, and that the Agency has embraced these two approaches, that is, by relying on animal test data to determine the chronic toxicity characteristics of various chemicals, and to add a large margin of safety by also relying on this one-hit, no threshold approach for setting toxic standards for certain classes of chemicals, especially those suspected of being carcinogenic in nature.

Will you please give us your views as to whether you feel these policies need to be changed, and if so, why should they be changed?

Dr. HERNANDEZ. Well, I think we are by and large still following that policy that you have identified. Each of EPA's statutory mandates has slightly different language in it, and to meet the test of the statute, you may do something slightly different, but by and large, in the absence of some other kind of information—that is, where your sole reliance is on animal testing—this is the process we follow today. Any kind of moving away from it becomes very, very difficult. I guess my lasting concern here is that we use the best information we have available and the total body of information in making decisions. But by and large, we are still using the same basic sort of approach.

Dr. ROWLAND. Are you doing this testing on what might be considered innocuous heavy metals, for example, lead? Are you doing studies on these types, also, not just those that you know pretty well are carcinogenic or do pose health hazards?

Dr. HERNANDEZ. Every year, we nominate compounds to go into a testing system that we share with other agencies of the Government. This is the National Toxicological Program. By and large, there is some kind of concern with those. Some of them turn out to be of no health concern, or not—we continue updating our data base in a number of different ways on chemicals, but as you would guess, our primary concern is those chemicals that, because of their chemical structure, we believe may be human carcinogens or toxic in some other way.

Dr. ROWLAND. Thank you, Mr. Chairman.

Mr. LEVITAS. Thank you very much, Dr. Rowland. Before we leave that question, Dr. Hernandez, do you have any particular feeling, professionally, or attitude as to the reliability or the reliance by the agency on environmental studies, epidemiological studies, as opposed to human test data?

Dr. HERNANDEZ. For the kinds of concentrations of environmental pollutants that we are looking at in many cases, it is impossible to have epidemiological data. America is a very mobile society. We do not stay put, and therefore, we do not stay exposed to these low levels over periods of time. And I believe that trying to find epidemiological case histories outside of the workplace, outside of some unusual exposure—the accident kind of thing—I think we almost have to rely on two things—one, the use of animal tests and other short-term tests that we have developed, but also on our understanding of the chemical and its mechanisms and its metabolites. I think that is what we have to rely on.

Mr. LEVITAS. I will get back to that later.

Mr. Wise.

Mr. WISE. Mr. Chairman, if you have no objections, I would like to trade my time with that of Mr. Albosta. He had some questions, and I thought it would be better if he were ahead of me.

Mr. LEVITAS. The gentleman from Michigan is recognized.

Mr. ALBOSTA. Well, I thank you very much, Mr. Wise. I appreciate this opportunity. This problem is in my district with Dow Chemical Co. and I think I have some very important questions to ask Dr. Hernandez.

Dr. Hernandez, was Dow Chemical Co. the only company that asked to see the report, or did they really ask to see the report?

Dr. HERNANDEZ. Well, I do not think I would have sent it to them had they not asked for it; and yes, they were the only company. Their name was in the report and in that newspaper article. You have to remember, that is what started this, was a newspaper article out of Canada, at a time we were trying to deal with the Canadians on acid rain and other kinds of issues, and Love Canal was in an initiative process. Yes, they were the only company that asked for it, and as far as I know, I never got anything in writing. I believe it was having seen Mr. Sercue in the hall. That is the best of my memory.

Mr. ALBOSTA. As I understand it, Dr. Hernandez, you have been a rather strong voice in the EPA and in the administration for good, strong scientific peer review.

Dr. HERNANDEZ. Yes, sir.

Mr. ALBOSTA. In your press release concerning this matter yesterday, you noted that the draft report had a thorough peer review by outside scientists——

Dr. HERNANDEZ. No, sir——

Mr. ALBOSTA [continuing]. And the report contained data which had been generated by Dow. And you say, "I felt that it was important that we get technical review from scientists involved in the generation of the original research."

Could you comment?

Dr. HERNANDEZ. Yes, I am not sure—I may have missed the first part of that. I believe I said that one of my reasons for being concerned about the report was that it had not gone through any rigorous peer review where someone, two people, are identified as having total responsibility to make sure that every fact in there is correct and substantiated in some way. It had not gone through that. It had gone through some kind of a committee process.

The second one in there was that some of the data was extracts from information Dow had made available through different processes, all of which, I am not sure—some of them through TSCA—abstracting what someone else said. When you have an opportunity to get their view of what they said, it is probably a better process. It is like that newspaper article quoted something else that I said. I gave them six items. They translated it in some other way. You read it back, and it becomes another thing. The best way to find out is to ask me directly, and that is what I said.

Mr. **Albosta**. That is what I am asking you.

Dr. **Hernandez**. That is what I said. Dow Chemical was the source of a lot of this information—its interpretation, the primary source is probably the best place to get whether or not that was a correct interpretation of the report.

Mr. **Albosta**. Was there any other data in that report that came from any other company?

Dr. **Hernandez**. Not to my knowledge, but that could easily have been. I have not read it for 18 months.

Mr. **Albosta**. Did all of the other literature and data sources or references listed in the draft have an opportunity to comment on the paper?

Dr. **Hernandez**. I have no way of knowing that, and certainly——

Mr. **Albosta**. Well, wouldn't a scientific review include all of those people?

Dr. **Hernandez**. No, not necessarily.

Mr. **Albosta**. Oh, then, what standard—how do you establish peer review in your department?

Dr. **Hernandez**. I can bring you a copy of the document. In fact, we can submit it for the record.

Mr. **Albosta**. Can't you briefly tell you whether you submit peer reviews to more than one scientific group?

Dr. **Hernandez**. More than one scientist.

Mr. **Albosta**. More than one scientific group.

Dr. **Hernandez**. Typically, you identify a scientist; you identify someone as being responsible.

Mr. **Albosta**. I would assume that you were trying to establish whether or not the facts in that draft or that report were accurate. How were you going to do that? That is what I want to know. What is your policy at EPA right now?

Dr. **Hernandez**. We have a well-documented policy of how peer review is done——

Mr. **Albosta**. Did you follow that policy in this particular case, then?

Dr. **Hernandez**. That policy was not in effect at this time.

Mr. **Albosta**. There was no policy in effect at that time?

Dr. **Hernandez**. That is correct.

Mr. **Albosta**. Well, would the gentleman yield—that is what I wanted to know.

What was the policy then?

Dr. **Hernandez**. There was no policy. That was one of the problems that EPA had suffered over the years, that people—scientists—had a lack of credibility in EPA's reports, because they did not reflect good, outside review of their documents.

Mr. **Albosta**. Well, would you consider good outside review submitting it to one company—and hadn't there been any peer review before that?

Dr. **Hernandez**. No. I do not believe.

Mr. **Albosta**. None?

Dr. **Hernandez**. On this issue, sir?

Mr. **Albosta**. No, not on this issue, on any other issues that EPA might have had before it. I am trying to establish, Dr. Hernandez, what EPA's policy was prior to your taking over.

Dr. **Hernandez**. The Office of Research and Development had a peer-review process. This is not a research and development document; it is a regional document, done by people out in the field.

Mr. **Albosta**. Don't you believe, though, that by deleting the material that was deleted there, that it did not give any particular significance to that particular draft or report?

Dr. **Hernandez**. Mr. Albosta, I had no idea what was deleted. I did not request anybody to delete anything. I have no substantive idea of why it was deleted. My involvement in this stems from two places—one, a discussion with the Regional Administrator on two occasions, I believe both on the phone, very short; and once or twice, perhaps, with some people out of the Dioxin Work Group, as to some issue—I do not remember at all what they were right now. And otherwise, people in EPA headquarters and in the region reviewed it, took the Dow comments into account where they were valid, I suppose.

You know, nobody has ever come back to me and said, "This was a wrong process. Why are we having to take Dow's word here?" Nobody ever said that. As far as I know, we did a good, open review of the thing and came up with a satisfactory report.

Mr. **Albosta**. Did you know that Dow Chemical Co. was withholding information for the last 1½ years from EPA pertaining to their studies on dioxin?

Dr. **Hernandez**. I have no knowledge about what Dow is doing and whether they are withholding it—does that mean that we do not get their stuff immediately? I do not know that, sir; clearly, I do not.

Mr. **Albosta**. What is the EPA doing, or what does it intend to do, pertaining to dioxin and the problems surrounding the Midland site, the problems in other parts of the country if they are there, in establishing strong, positive, scientific evidence on whatever level of dioxin is safe for people? I am concerned about the people I represent in my district, and I want to know that everything is being done by Government agencies that are paid and have been authorized and legislated to do certain things through appropriation bills, to see that this area of safety is carried out. I am concerned about that, and I do not feel that everything is being done, and I want to know why, and I want to know what is going to happen in the future and what are going to be your recommendations on this very problem that we are faced with here today.

We go from one side of the coin to the other, and we cannot straighten this thing out. EPA's record and its integrity has gone down the drain; it is down the Tittabawassee River.

Michigan has asked for money to do studies. Is EPA going to give that money to the State so it can complete its studies—$495 million, I believe?

Dr. **Hernandez**. I have never seen a request, but we will certainly consider whatever is appropriate for the State of Michigan. Let me tell you some of the things we are doing on dioxin.

This report covers a lot of things other than emissions that are purported to be from the Dow Chemical plant. This report covers items like the emissions of dioxinlike materials from waste combustors. We have conducted five studies, maybe six studies now, on waste combustors, to determine whether or not the levels of dioxin emissions from these things are at levels that may be harmful to the health of people living downwind. That is one of the concerns raised in this report. Each one of those studies cost us on the order of $150,000 to $200,000. We are making a major investment in that area, and we are going to continue to look at that.

Other places where dioxins are being investigated at breakneck speed is in that whole Missouri area. We have taken literally hundreds of samples out there of areas where they are thought to be contaminated with dioxins. Any idea that we are not pursuing this with great vigor just has to be a misunderstanding. The amount of money we spend in that Missouri area and the involvement of our people, I really believe——

Mr. Albosta. Let me ask you another question. When you submitted that to Dow, did you know that Dow had refused to cooperate with EPA and as a result that the Justice Department has intervened concerning their reluctance to provide dioxin data?

Dr. Hernandez. I have no knowledge of that at all.

Mr. Albosta. You have no knowledge of what is going on over at the Justice Department pertaining to dioxin?

Dr. Hernandez. No, sir, I did not say that. I said that this particular case, with respect to Dow and the Justice Department, I have no personal knowledge of. Could we supply that for the record, because if it is an enforcement issue with EPA, I think we will probably find it. You are characterizing it as not providing information; I have no idea about that.

If you could give us the particulars of that, we would be glad to give you that for the record.

Mr. Albosta. Certainly, we will see that you get the particulars. Let me say, Dr. Hernandez, I would like to have you——

Mr. Levitas. Excuse me, Mr. Albosta. Would you just yield at that point, because before you leave it, I think perhaps either Dr. Hernandez or someone else from EPA here might be able to provide you with the information you are looking for.

I think Mr. Albosta was referring to something that is of concern in this instance of dioxin. It is my understanding that at or about the time you gave Dow a copy of the dioxin report to review, that Dow was at that time and still is refusing to give the State of Michigan and EPA their own data on dioxin discharges, and that this Dow dioxin data is being sought pursuant to NPDES permit application.

Dr. Hernandez. I am sorry. I do not think anybody here—it is an NPDES permit [conferring with staff] we have regional counsel here. Would you like to swear him in, and we will get a firsthand report on it. I think we can——

Mr. Levitas. All right. I do not want to intrude on your time, Mr. Albosta, but I think this is what you asked for.

Mr. Albosta. Yes, I appreciate you spelling this out in the technical terms.

Mr. Levitas. Does anybody have that information?

Dr. Hernandez. Yes, sir.

Mr. **Brown**. One of our attorneys from the region V Chicago office is here, and his superior, the Regional Counsel, tells me he has some information on that.

Mr. **Levitas**. All right, would you please come forward?

Let me administer the oath to you, please. Do you solemnly swear the testimony you will give before the subcommittee today will be the truth, the whole truth, and nothing but the truth, so help you, God?

Mr. **McPhee**. I do, sir.

Mr. **Levitas**. Thank you. If you would take a seat and identify yourself by name and title, and provide us whatever information you can on that point.

Mr. **McPhee**. Mr. Chairman, my name is John McPhee. I am an attorney in the region. Up until a few months ago, I was assigned to the matter that I believe is being discussed here, and I will provide whatever information I can. I would like to have a clear statement of the question, though, if I could.

Mr. **Levitas**. The question is whether or not Dow Chemical Co. has refused to provide information with respect to dioxin discharges in Michigan that is being sought in connection with an NPDES permit application, and that that refusal was in existence at or about the time the report we have been talking about was provided to Dow, and what is the status of that?

Mr. **McPhee**. Beginning in early 1981, we made a request to Dow Chemical Co. under section 308 of the Clean Water Act, for information about several kinds of activities at the Midland plant. We wanted a characterization of the wastewater flows from the plant. We also wanted process information about those processes that might be discharging a wide variety of toxic substances.

There were negotiations for a substantial period of time with Dow that were unproductive to a certain extent. Some information has been provided to EPA about certain aspects of the discharge from the facility. During this period of time, we also engaged in a study ourselves, to try to determine certain information about the kinds of materials that were discharged from the facility.

There are several remaining areas where Dow has declined to provide information that we requested and we thought was necessary and important to the permitting function under the NPDES process.

The matter was referred to the Department of Justice for litigation and there is a case presently on file.

Mr. **Levitas**. And is one of those substances dioxin?

Mr. **McPhee**. Yes, sir.

Mr. **Levitas**. Mr. Albosta.

Mr. **Albosta**. Dr. Hernandez, are there rivers and lakes in Michigan contaminated with dioxin, and if so, at what levels; and what is the danger posed to the residents—tell us that?

Dr. **Hernandez**. I think I can answer part of that; part of it, I cannot. Let me ask our lawyer from region V to just provide some dates on when these things happened, when it went to litigation and so on, if he could, on that issue.

Mr. **McPhee**. The case was filed on the 15th of December, as I recall, of last year—1982. There were, as I say, negotiations over a long period of time; a lot of back and forth on the subject, or a number of claims and defenses that are probably going to be raised in litigation. The important date, I guess, is the date of filing of the litigation, which is the 15th of December. The other important date would be the fact that we did serve this interregional information request on them in January 1981, and were not successful in obtaining the information, in toto.

Dr. HERNANDEZ. I think I would go back to the other question.

Mr. ALBOSTA. Certainly. I want to know what health hazards are posed by this. You have got to have some idea. There has been scientific review, I believe, that is simply very clear that it is the most toxic chemical known to man. Is that true—in your opinion?

Dr. HERNANDEZ. I believe that the 2,3,7,8 isomer of TCDD is probably the most toxic chemical that we have. There are 75 so-called dioxins, not all of which have anywhere near that toxicity. So that particular isomer is very toxic.

Mr. ALBOSTA. What is the lowest one? What would you compare it with—gasoline, something else? Tell us what the lowest one would be.

Dr. HERNANDEZ. Well, I really hate those kinds of comparisons, because you have people trying to compare the chances of smoking two packs of cigarettes a day and the risks you run vis-a-vis some of these others, and I believe it gives society some kind of an unbalanced view of the risks.

Mr. ALBOSTA. Well, from your scientific knowledge, Dr. Hernandez, you would have to have some idea. Is the statement that dioxins are the most toxic chemicals known to man—dioxins—they are talking about them all when they say that, I would assume. In your point of view, is that so—not only with the highest one, but with the lowest one?

Dr. HERNANDEZ. That is why I was trying to clarify that issue, that the principal one, the 2,3,7,8, is probably the most toxic chemical that we deal with in our society out here, that is found frequently in places.

Now, there are other dioxins that are found. Characterizing them against each other is a difficult lab technique, but do-able.

Now, you ask what is the problem of eating fish out of these places. I think each one of those has to be assayed on the water from which the fish are taken, the types of fish we are talking about, the likely frequency with which somebody might consume that particular fish—that is, is it a year-around fish, or is it a summertime sportsfish, or is it a commercial fish. So, making statements as to the relative safety——

Mr. ALBOSTA. So you would rather that it all be withheld and not say anything, just let them be the guinea pigs, and let them die, and if they die, then we will make a determination afterward? Is that what you are telling me?

Dr. HERNANDEZ. I am not telling you anything like that at all.

Mr. ALBOSTA. Well, what are you saying, then?

Dr. HERNANDEZ. I am telling you that on each one of these, you have to make a very specific and concise statement, and I am not going to give you some kind of an offhand——

Mr. ALBOSTA. But you are saying it is OK to eat the fish.

Dr. HERNANDEZ. If you ask me the name of the fish, I will tell you.

Mr. **Albosta**. I'll name a sucker. Suckers are caught in those rivers.

Dr. **Hernandez**. I am going to have to ask a question to some of our staff who may know about that. May I, for a second? I do not know anything about suckers, particularly.

[Laughter.]

Dr. **Hernandez** [conferring with staff]. We are going to take a look in the report for suckers. The FDA—let me tell you how we establish whether or not——

Mr. **Albosta**. Let me say this. If you have a fish in there besides the sucker, let me know which one it is. If they are in our rivers, and they should not be consumed by our people, I want to know about it, and I want this committee to know about it. And I want EPA to let everyone in our area of Michigan know about it. I do not want, 10 or 20 years from now, people dropping over dead, and EPA coming back and saying,

> Oh, my God, it is too bad we really did not make a decision at that time that dioxins were that toxic, and we should have advised the people, but we did not. It is tough, you are dead.

Mr. **Molinari**. Would the gentleman from Michigan yield?

Mr. **Albosta**. Yes, I will yield to the gentleman from New York.

Mr. **Molinari**. Dr. Hernandez, in the original draft report's, introductory remarks at the top of the page, you discuss talk trace contaminant, 2,3,7,8—tetra-cholro-dibenzo-p-dioxin, TCDD, saying that "This chemical, commonly known as dioxin, is possibly the most toxic chemical synthesized by man."

Dr. **Hernandez**. That is right. That is consistent with what I said.

Mr. **Molinari**. It is interesting to note that in the final report, that language was changed somewhat, and instead, you described that chemical compound as being the most toxic of the 22 possible isomeric forms of TCDD.

Dr. **Hernandez**. Both of those statements are correct.

Mr. **Molinari**. Why the change?

Dr. **Hernandez**. You know, scientists—and that is who made these things; scientists made those decisions; I did not make those decisions as to word changes. There were a large number of people who had input from within EPA—competent people who provided that kind of background.

There were no policy decisions on how those words were written down, believe me.

Mr. **Molinari**. I do not want to preempt the gentleman from Michigan's time, but this subcommittee in the very near future is going to be going to Missouri to do a field hearing on the dioxin problem out there. It seems to me that Mr. Albosta has been trying to get an answer on how serious this dioxin question is in relation to the other problems that your agency has dealt with.

Dr. **Hernandez**. OK. Let me just say one thing, so that you cannot say that we did not tell you. The report that you have a draft of up there; the final version went to a number of people in July 1981 from EPA. It was finished and sent out. It contains these levels of dioxins in fish in Michigan rivers, and there is the full weight of the report that says, "Here is the kinds of levels that we obtained. There is an FDA level to compare that against," and FDA takes into account that long-term consumption and all of the other things. They are the ones who decide what is safe in food. And looking at the fish in these different waters, the State of Michigan has the responsibility of deciding whether or not to ban fishing for that type and commercial fishing and so on. And I believe that this information was made available, so that that information is available. We are not withholding; we are not waiting until people die in the streets.

Mr. ALBOSTA. I believe that Michigan did run some tests, and they ran them near the outlets of Dow Chemical Co.'s fluid that is going into the river. And I am not trying to single out a company in my area, and I want you to know that if I were Dow Chemical Co., I think I would have probably done the same thing they did.

What I am trying to single out is your agency. Your agency is the one that I do not believe has operated properly; not Dow Chemical Co. The things that happen with toxic chemicals in many cases can only be prevented if we have a proper agency to regulate. But if they are going to provide coverups or if they are going to provide the opportunity for that, then I do not blame the company.

Do you think that Dow acted properly or improperly in this matter? What is your own personal opinion?

Dr. HERNANDEZ. Would you mind separating that in terms of concern over the initial report, or not providing information to us; that is the question?

Mr. ALBOSTA. The initial report—leading from the initial report.

Dr. HERNANDEZ. They did not delete.

Mr. ALBOSTA. Who did?

Dr. HERNANDEZ. The people who finally wrote and published the report, the people in region V. Dow Chemical did not delete anything.

Mr. ALBOSTA. How did it get deleted by region V?

Dr. HERNANDEZ. That kind of review process, that copy I gave you, has all kinds of markings on it from the May review internally, in-house; it has those critical comments on the front side of the package. Those reviews went on. Those reviews went on because of some of the concerns that I had raised.

Mr. ALBOSTA. But where did the recommendation come from?

Dr. HERNANDEZ. From staff at EPA, my concerns.

Mr. ALBOSTA. It did not come from Dow?

Dr. HERNANDEZ. Dow Chemical, I believe, made some inputs to our staff. I had no idea when, in what form what comments they made, at all. Those were not transmitted to me. I had no way of telling what they were. I did not tell anybody to take their word for it and to put them in there.

Mr. NOWAK. Would the gentleman yield?

Mr. ALBOSTA. I yield to the gentleman.

Mr. NOWAK. You had mentioned several times that you had no input, Dr. Hernandez, into the changes that were made in these documents. I wonder if you could tell us the names of the people who were involved in making the requested changes submitted to them by Dow Chemical?

Dr. HERNANDEZ. I would have to, if I could, ask one of our people from the Chlorinated Dioxin Work Group to supply some of those names. Could we do it for the record—or, we could give you some of them now. I think some of them are on the package.

Mr. **Nowak**. We would certainly like them for the record, I would believe, and if you could supply as many as you can at this point in time, and you could supplement that later with a full record statement.

Dr. **Hernandez**. I believe that all of our Chlorinated Dioxin Work Group, which is a very large body—I think it is on the order of 20 people—had input into this. They come from all over the agency, all different programs.

Mr. **Nowak**. Well, wasn't there a chairman of that input group?

Dr. **Hernandez**. Yes, there were two people. I think Paul Brown, whose name appears, and I think Dr. Don Barnes was the other cochairman of that group.

Mr. **Nowak**. They would have been the people who would have taken the draft back and examined the deletions which were requested, in my understanding, from Dow Chemical Co.?

Dr. **Hernandez**. Not necessarily, and I do not know where those went to or how they got there, whether they came in written form, or what.

Mr. **Nowak**. I wonder if you could also supply that to the committee——

Dr. **Hernandez**. How that back-and-forth went, certainly.

Mr. **Nowak** [continuing]. As to how the comments were put into the draft from either written request from Dow, oral conversations, and who was involved with those oral conversations.

Dr. **Hernandez**. We will try to reconstruct all of that. It is 18 months ago, and when you do not have these things raised, people forget about all the input. This has newly come as some surprise to us.

Mr. **Nowak**. Then, the person who was in charge of that final draft, could you identify him?

Dr. **Hernandez**. I believe, the man who authored it; I am not sure his name appears. Hang on. The original one was written by Dr. Clark. The final draft does not have his name on there. The people in region V, in their toxics branch, would be the editors and final authors of this, and it is not a bad piece.

Mr. **Nowak**. Well, that seems to be open to question. All I am trying to do is find out who would have allowed the draft to be changed.

Dr. **Hernandez**. Allowed the draft to be changed—the authors.

Mr. **Nowak**. You said that Dr. Clark was the first author.

Dr. **Hernandez**. I guess he works in that group there.

Mr. **Nowak**. All right. Now, did he approve, then, the requested changes by——

Dr. **Hernandez**. I have no idea. I would guess he did. I never heard from any of the scientists there, or any of the law enforcement people, or anyone that there was anything irregular or unprincipled about this at all.

Let me make an offer, and it goes like this. If there is anything in the original draft that is sustainable by the facts in that draft that do not appear in here, in the final draft—which came along very quickly thereafter, within 15 or 20 days, so there was not a lot of time for new information—then, I must be at fault, because I really believe that anything that we had that was good, basic information that contributed to the report on this subject should have appeared in the July draft.

Mr. **Albosta**. I see my time has expired, and I want to be fair, Mr. Chairman.

Mr. **Levitas**. Thank you, Mr. Albosta. We will get back to you, because you are pursuing an important line of questioning.

Mr. Sundquist?

Mr. **Sundquist**. Thank you, Mr. Chairman.

Dr. Hernandez, would you say in retrospect that the way you handled the whole Dow question was bad judgment?

Dr. **Hernandez**. No.

Mr. **Sundquist**. Did you make any errors in judgment at all in that decision?

Dr. **Hernandez**. Yes; but let me tell you——

Mr. **Sundquist**. If you made errors, then why wasn't it bad judgment?

Dr. **Hernandez**. Well, let us look at the overall product that we got out. We turned out a credible report that does the basic job that we wanted to establish the kinds of sources in the Great Lakes, the sources of dioxin, and we did it in a very timely fashion without a lot of controversy after these problems were reported, without any policy statements in it, and any——

Mr. **Sundquist**. I am not referring to the quality of the document. I am referring to leaving yourself above reproach which you did not, apparently.

Dr. **Hernandez**. Well, decisionmakers have to make decisions every day——

Mr. **Sundquist**. I understand.

Dr. **Hernandez** [continuing]. And they make them on imperfect information, and I am going to go on making them, and I am going to make some mistakes, and second-guessing me is the easiest damned thing in the world.

Mr. **Sundquist**. Why is it, then, that you say you have made policy correction and that this will not happen again?

Dr. **Hernandez**. Well, I think we have put into effect a good peer review process now that would—not prevent it totally; you never do that—but help preclude it by providing a process for internal review and, where it is appropriate, external review by scientists, and we do this all the time.

Mr. **Sundquist**. So what you have, then, is a program to prevent this kind of thing from occurring again, is that correct?

Dr. **Hernandez**. For trying to solve all of these kinds of problems of how to deal with these complex technical reports that——

Mr. **Sundquist**. Well, if you made a mistake, just say you made a mistake.

Dr. **Hernandez**. Well, I would tell you that I make lots of mistakes in my life. Let me just make it clear where I believe my mistakes lie. The overall outcome was a good outcome. It is a report that does not have conclusions that cannot be sustained by the information in them. Now, that draft report did.

Mr. **Sundquist**. I understand.

Dr. **Hernandez**. The outcome was a good outcome. Now, whether or not I put myself liable here, 18 months later, to criticism, well, that is just the breaks of the game. Now, one of the things I probably should have done was to have said, "Let's send this thing out to the world, and I will cover myself, then, by sending it not only to Dow, but sending it to everybody else, and that will bless the process." And I probably will do that in the future, if we ever do another one like this.

Mr. SUNDQUIST. Do you plan on reorganizing or making any management changes as the Acting Administrator at EPA?

Dr. HERNANDEZ. Oh, I think there will probably be some minor reorganizations. It depends a lot on time. But I think we will look at some very minor ones to cure some problems. Management, yes, I think each of us through our own characteristics and our own background have different management styles, and certainly, there will be changes there.

Mr. SUNDQUIST. As the top ranking scientist within EPA, what in your opinion are the most critical problems that we face 10 or 20 years from now in terms of the environment?

Dr. HERNANDEZ. I am thinking about that.

[Pause.]

Dr. HERNANDEZ. You know, I think we will probably still be in that kind of mixed system 10 or 15 years from now, where there will remain uncertainties about human exposure to low levels of chemicals, and I think that probably will remain one of our great concerns. You know, the more we learn about some of these things, the more difficult it is to do decisionmaking. And I believe that we will still be thrashing around with some of those problems.

Mr. SUNDQUIST. I am concerned about EPA creating new Love Canals. In light of the fact that the state of the art set previously allowed Love Canal to occur. According to the state of the art now, EPA approves dumpsites based on certain criteria or narrow requirements but not whether the dumpsite is needed or not.

I am told that there are two EPA-approved dumps that are presently leaking. Is that correct?

Dr. HERNANDEZ. Two that are leaking?

Mr. SUNDQUIST. Yes, one in Indiana and one in Colorado.

Dr. HERNANDEZ. I cannot speculate on that. I do not know——

Mr. SUNDQUIST. I do not think that is speculation. Either they are, or they aren't.

Dr. HERNANDEZ. But I am saying I do not know about them.

Mr. SUNDQUIST. Does any of your staff know about them?

Dr. HERNANDEZ. It would take a quick call.

Mr. SUNDQUIST. Thank you.

Dr. HERNANDEZ [conferring with staff]. A quick answer. We have not permitted any land disposal sites, so there are not any that are EPA approved.

Mr. SUNDQUIST. In either Indiana or Colorado?

Dr. HERNANDEZ. Anywhere.

Mr. SUNDQUIST. You have no EPA-approved dumps in the country?

Dr. HERNANDEZ. No, no. There are some State approvals.

Mr. SUNDQUIST. All right, State approved.

Dr. HERNANDEZ. There are none that have a permanent EPA permit. There are some on interim status right now.

Mr. **Sundquist**. I do not want to get technical, and I do not want you to get technical. I am not an attorney, I am a businessman, so I would like to have you be as direct as you can. Even though they are approved by a State and may be on an interim-status basis are there some state-of-the art dumps that today are leaking?

Dr. **Hernandez**. You know, I am going to say it is a real possibility that there are some that will leak. Part of the state-of-the-art provides for back-up systems of monitoring. That is why you have these multiple systems of, first, trying to prevent leakage, and then, secondly, trying to detect it if it occurs, and that that kind of monitoring process be so close to the site that you can control problems before they move far down the line.

Mr. **Sundquist**. Are some leaking?

Dr. **Hernandez**. You know, I am going to say that it is a probability that that is the case. Certainly——

Mr. **Snyder**. Why don't you let Mr. Lucero answer those questions? He is in enforcement.

Dr. **Hernandez**. My guess is you are right, heck, yes. Could we put Gene Lucero in, too?

Mr. **Sundquist**. I do not need a long answer, Mr. Lucero.

Dr. **Hernandez**. This is Gene Lucero.

Mr. **Snyder**. He has been here before.

Mr. **Lucero**. That is right.

There are a number of facilities—I would like to clarify one thing, quickly. What Mr. Hernandez said was there are no permitted facilities, and that is because EPA is embarking on the process of permitting either directly or through the States, under the new RCRA land disposal regs, those facilities. Now, those facilities that met the requirements for interim status and were permitted to operate in the interim period from November 1980, there are some facilities that have shown leaking, and we are at those facilities dealing with both the inspections and enforcement.

Mr. **Sundquist**. Thank you. That was my question.

Based on the fact that we have this problem, shouldn't EPA be focusing on whether or not there are some alternatives to landfill and the fact that we are still putting things in the ground that are going to create problems in the future?

Dr. **Hernandez**. I certainly agree with you on that, and we are. There are a number of processes, both in the solid waste organization and in research and development that are ongoing in that area.

Mr. **Sundquist**. Do you or your staff intend to come back before Congress and say the legislation that you passed needs some adjusting to correct these kinds of problems, or, that you need more money. In other words, EPA's responsibility is to correct the problem. The public does not have the confidence that EPA is correcting the problem. What are you going to do to restore confidence, what are you going to do to get things moving, what are you going to do to communicate, so that you are not above reproach? Whether you agree or not, I think your judgment on Dow Chemical was inappropriate. I think whenever the Agency's action is questioned in case like this, the Agency is injured and the whole environmental question is injured.

Dr. HERNANDEZ. You know, I agree with you. But if we waited around to make decisions every day on the basis of some of these things, the unknowns, we would not do anything. I made a decision. It has turned out that, now that I have become the acting administrator, it has become a very big issue, and people have focused on it.

Mr. SUNDQUIST. Well, what about the legislation question? Are you preparing any legislative proposals for Congress that will help make your job easier and more effective?

Dr. HERNANDEZ. Yes, sir; in a number of areas. We hope to have some RCRA recommendations to the appropriate committee, and certainly, the Clean Water Act, we have got some coming along in the process. So the answer is yes, and if we need more money, we will be coming back to the Appropriations Committee with recommendations.

Mr. SUNDQUIST. Would you give me the capacity of chemical dumps in this country and convert that into years of usage that we have remaining in those dumps. I do not need it this minute, but I would like to have it from EPA.

Dr. HERNANDEZ. And you are talking about ones that are either permitted or interim permitted——

Mr. SUNDQUIST. Correct.

Dr. HERNANDEZ [continuing]. So that we have some chance to——

Mr. SUNDQUIST. The question is, Do we need more dumps right now, or should we wait a couple of years to see how things work out?

Dr. HERNANDEZ. Clearly, Mr. Sundquist, one of the real problems identified in my opening speech was the need for sites for disposal of these wastes. I said that was going to be one of the——

Mr. SUNDQUIST. If you will tell us how much capacity we have, or if you know how much capacity we have, we may not need more sites.

Dr. HERNANDEZ. Yes, sir.

Mr. SUNDQUIST. Thank you.

Thank you, Mr. Chairman.

Mr. LEVITAS. Thank you, Mr. Sundquist.

Would you submit that information for the record?

Dr. HERNANDEZ. Certainly.

Mr. LEVITAS. Without objection, it will be received.

The gentleman from Texas, Mr. Andrews.

Mr. ANDREWS. Before the recess this morning, Mr. Molinari was following up on a line of questions with you about moving resources around in a particular region where they were not prosecuting enough cases or did not have enough cases, where funds were withdrawn from that, and I am not clear on your response to the tenor of his questions.

Do you agree with that policy, or do you disagree with that policy?

Dr. HERNANDEZ. Certainly, you have to look where you have problems and put your resources there. Some kind of arbitrary approach is certainly not a valid one. And I think that he was implying that this was an arbitrary sort of approach to dealing with something that was much more complicated than that. And clearly, reason would say where you have problems, you put people.

Mr. ANDREWS. So your response is that you disagree with that policy?

Dr. HERNANDEZ. I think you have to look at it in the overall context of how it was put forward, at what time, for what purpose, and what else was with it. Now, taking that out of context is difficult to deal with, Mr. Andrews. You have to look at what the overall piece was. If it was an arbitrary action of, "If you do not produce, we will take back part of the marbles," that becomes a very negative kind of incentive. What we need to do is look——

Mr. ANDREWS. And you would disagree with that?

Dr. HERNANDEZ. What we need to do is to look for the places we need resources and put them there. And that is what we hope we would do.

Mr. MOLINARI. Would the gentleman yield?

Mr. ANDREWS. Certainly.

Mr. MOLINARI. Have you concluded with that line of questions?

Mr. ANDREWS. Yes.

Mr. MOLINARI. When I did ask that question earlier today, Mr. Crampton nodded his head rather vigorously. I expect that he wanted to offer some words of advice to us on that and, besides, you have been very quiet today. So, would you mind giving us the benefit of your thoughts on the subject?

Mr. CRAMPTON. Thank you very much, Mr. Molinari.

Basically, this initiative from Mr. Perry's office, you have to understand, came about in the context of the Agency taking a lot of criticism for not litigating cases, and as you recall, there was a lot of talk in the press about how slow we were to enforce. And I think that Mr. Perry, in response to those criticisms, was moving on his own to try to deal with the problem. Now at the same time, there were considerations over in the Program Office about favoring administrative actions for enforcement, administrative orders under section 106, and notices of violations, and other sorts of things. So Mr. Perry's letter sort of caught that effort kind of in midstride. What our office did—which is the Evaluation and Accountability Office—was advise the administrator that there was indeed a conflict here between the legal people and the program administrative people, and we advised her as to how this particular issue might be worked around, and in fact, it was. When the discussions with the budget people over the workload models were entered upon, this matter was taken care of, a new set of criteria was sent out to the regions, and we worked it a totally different way.

So you caught a memo that created some flack for a while, but it eventually got dealt with within the system correctly, I believe.

Mr. MOLINARI. That is the first time we have had a reasonable explanation of what transpired following the issuance of that memorandum, and I am grateful to you for giving us that explanation.

Mr. ANDREWS. Thank you very much for your comments, and that brings me to another question I have for you, Dr. Hernandez. Just in terms of internal philosophies, the disagreements between those who wanted to go ahead with prosecution—in many instances, Mr. Perry—and those who felt it was necessary to negotiate first with polluters, I would like to know your philosophical view about that—and specifically, there was a memorandum passed around within the Agency that said negotiations should go on first and then litigation. That troubled me, and it troubled other members of this committee, that in fact, the prosecutorial discretion would just be somewhat undermined if the polluters out there knew that the EPA's attack would be to simply sit down at the table and start negotiating, and that you may lose what leverage you would have as an adversary—leverages that you would have with the imposition of treble damages, and the costs that litigation would mean not just to the United States, but to the polluters themselves, in terms of helping you down the road reach a settlement that is satisfactory to the people in the community and, as importantly, the U.S. Government. I wonder if you would comment on that philosophy, that was really proffered to this committee by Ms. Lavelle and what your view is.

Dr. **Hernandez**. I think that is a really good question because it becomes a fundamental one. Let me say it is one that we have talked about very briefly since my becoming the Acting Administrator, and one that I have not thrashed out totally, but let me give you my sixth sense right now, and likewise, I think it is not inappropriate to hear some comments back from you; you have expressed one.

I think the first thing that we need to do in each of these cases is the investigation piece, so that we know what is there and what our estimates are of what it is going to cost, and that is pretty well done when you get down to that feasibility stage. So, the first part of this should be an information-seeking thing. At the same time, the enforcement people probably ought to send those letters out and say, "We believe that you were associated with this site; come forward," and whatever those letters are that put people on notice, so that as you move down, you have the kinds of opportunities and to make decisions on a case-by-case basis, based on some things you would look at. If it looked like you might be able to come to a quick—because these people are going to be coming forward and saying, "Listen, let's talk about this"—and the feasibility studies are going on, and those take some time. We might get a guess from Mr. Hedeman how long, but my guess is, even on an uncomplicated site, you are looking at a couple of months' worth of time, three months' worth of time, after you get started on it. So, moving down the line, you ought to be able to make those decisions on whether or not you can come to quick settlement with these people; if you cannot, we should immediately start that other process of making decisions on us doing the cleanup, and we will take them to court to collect whatever the court finds is appropriate, which could be up to the treble damages.

Mr. **Andrews**. Do you disagree or agree with that statement, that the EPA should negotiate first and then litigate? Do you think that is an appropriate tenor of procedure, or policy and procedure within the Agency?

Dr. **Hernandez**. It is too simplified. It is too easy to characterize as this piece. What I would like to see is a little more complicated kind of policy that looks at steps as you go down the line, and you get to this point, and then you make that decision.

Mr. Andrews. Let me ask you one more question along that line. Where there were disagreements within EPA between Mr. Perry or Ms. Lavelle as to whether or not a case should be settled or prosecuted or negotiated, at what stage settlement negotiations should start, there appeared to me to be almost a total impasse at times between what Mr. Perry thought was appropriate and what Ms. Lavelle thought was appropriate.

What specific steps do you plan to undertake within the Agency where you have disagreements or judgment calls, prosecutorial discretion, that are total divergent within your Agency? Now, specifically, do you intend to carry out what you think the goals of the Agency should be?

Dr. Hernandez. Mr. Andrews, in all candor, I really have not thought that one out that clearly. I think that we will either do it through experience with some mistakes, or perhaps there may be some kind of case history on what is an appropriate kind of thing. I just haven't the experience at this time to give you——

Mr. Andrews. Who will make the final decision?

Dr. Hernandez. I will.

Mr. Andrews. Thank you.

Mr. Levitas. Thank you, Mr. Andrews.

Ms. Johnson.

Ms. Johnson. Thank you very much, Mr. Chairman.

I just have a couple of very brief questions that go to an aspect of the Superfund legislation that concerns me very much. Your prepared statement quite clearly outlines some of the problems from the States' perspective of implementing the Superfund Program. Among those is the cost-sharing requirement. Do you have any reason to believe that the cost-sharing requirements may have influenced States' decisions in the selection of nominated sites for the national priorities list?

Dr. Hernandez. I think that is a really reasonable question. I do not have any, personally, but could I ask Mr. Hedeman to answer that one?

Ms. Johnson. Yes.

Mr. Hedeman. No, Ms. Johnson, I do not think they did.

Ms. Johnson. You have no evidence that cost influenced their willingness to make those decisions.

Mr. Hedeman. The specific question was in terms of nominating the sites to the national priorities list, and my answer is that we implemented a policy to the effect that we would establish on the national priorities list all sites that we knew to be the most serious, regardless of whether the States agreed with us or not, or were willing to submit the site for nomination. There are a number of sites that are on the national priorities list that are on there without the concurrence or consent of the States.

Ms. Johnson. I recall that from your earlier testimony, and that helps to put this in perspective. In proposing amendments to the Superfund legislation, will you be addressing the problem where many of our States require independent legislative action on a site-by-site basis, or do you consider that a problem?

Mr. Hedeman. I think it is a problem, and we are at this very moment, taking initiatives to provide recommendations to the

States as to how they may be able to solve that problem within their individual institutions.

Beyond that, I do not think we are in a position to give any recommendations at this point. Could I give you examples?

Ms. JOHNSON. Yes.

Mr. HEDEMAN. We see three general approaches that States can take now without having to consider changes to the law, in order to come up with the prerequisite 10-percent or 50-percent cost-share. One would be to use the existing assets within the State to generate new revenues. An example of that would be that, as we have studied many States, we have found that there are existing funds or revenues within the State to respond, for example, to emergencies, but the moneys are not being managed in a way to collect any interest on the funds, and so the States simply sit there with an account of money that is generating no interest from one year to the next and being supplied annually by additional appropriations.

Another example is to capture what we call economies of scale. For example, many States in other programs have entered into agreements with sister States and/or with industries to copurchase or comanage equipment necessary to clean up sites or to respond to emergencies so that we do not have an overduplication of effort, so to speak, most of which is sitting around, waiting to respond to a certain event. The third one would be to accelerate the cash flow in the fund, in existing State funds, by, for example; using moneys that have been dedicated for emergency or hazardous waste problems within a State, to respond to the 10-percent requirement, and taking the money that has been appropriated to the State for emergency purposes, and perhaps entering into insurance arrangements with Lloyd's of London or some other insurance company, so that the money within the State can be dedicated to the 10-percent match.

We have worked toward a draft document, or guidance document to this effect, that gets much more elaborate than I have with you——

Ms. JOHNSON. I do not feel it is necessary to go into further detail. I appreciate the kinds of things that you are willing to talk with the States about and your willingness to evaluate their resource management. It does concern me that many of our States have a site-by-site process. As an old State legislator, I know how that can delay action. I think especially in the Superfund area those kinds of delays may be simply intolerable from the point of view of public health and safety. I am interested in the experience that you have accumulated to this point, to at least provide some backup to strengthen and enhance implementation of the law which is of such enormous importance, not only, possibly, to that State, but to the citizens of the adjoining States.

Mr. HEDEMAN. Well, we are, and our approach, frankly, would be to use the services of the National Governors' Association or similar groups, to bring the States together and share with them our suggestions of how they may proceed.

If I could take 1 minute to follow on to that question, I think the subcommittee may find of interest the problems we are having in this area with respect to Missouri and dioxin.

When I testified 2 weeks ago, I indicated that the permanent relocation offered at Times Beach in Missouri was one in which, while we had made money available, we could not fulfill that commitment until the State of Missouri came forth with the 10-percent match. That has yet to occur. The State Senate reported out yesterday or the day before an appropriation of $3.5 million, with a proviso, as best as we can determine, that the Federal Government own the land; in other words, that when the houses are purchased, the land would be owned in the end by the Federal Government. That puts us ~~in an~~ interesting catch-22 situation, because if we wind up owning Times Beach, we are precluded by the law from taking action on Federal lands, and action would be correcting the problem. So we get into this gamesmanship.

Aside from that issue, if I can focus the problem further on another site in Missouri—we call it the Minker-Stout site, and it was on Nightline" last evening as a site, in fact, one of the first ones which received in a residential area a large quantity of fill material containing dioxin-contaminated oil. We have done some preliminary cost estimates at that one site, to clean up just what we call the Minker-Stout area, and in that context, based on our preliminary determinations of the extent of the contamination, we find that roughly 5,000 to 8,000 cubic yards of soil would have to be removed. In layman's terms, that is 800 dump trucks. One approach would be to construct a grout curtain around the site, which is bordered on both sides by two streams which are heavily contaminated with dioxin waste. To construct that curtain, which would take about 4 years to construct, would cost approximately $29 million. That would require using, among other things, an estimated 100 people in full protective gear to handle or be in the vicinity of the area while the material is being moved, so that they are not exposed to the contaminated soil.

A second approach is to remove all the soil and place it in a vault, a concrete vault that could accommodate 5,000 to 8,000 cubic yards of material—again, $9 to $11 million, 2½ to 4 years to construct the vault and put the soil in it, and frankly, find a place to put the vault in the first place.

The third one is to dig the soil up, which seems to be relatively simple, put it into the 800 dump trucks and take it to a licensed disposal facility. There are no licensed disposal facilities, at least in the State of Missouri, for dioxin-contaminated soil, and probably others that may exist within the States are unwilling to receive such a huge quantity of soil because of the capacity involved. If we can go that approach, our estimates are that it would cost $4.5 million to $8 million to truck this soil across the State of Missouri to wherever it is headed, and also the related cost of preparing the trucks to ensure that the soil does not fall out en route, and dealing with the citizens' concerns in the transport.

The fourth one is to incinerate the soil. This is a process that the State has advocated and we have been willing to discuss with them. If we proceed in that direction, it will take 8 years to incinerate the soil at the Minker-Stout site and cost somewhere between $29 million and $38 million. That is assuming that that process can work, and then you have the problem of the residue that is left; that has to be put someplace as well, because there is still contaminated soil residue of dioxin that would have to go to a licensed disposal facility.

And finally, the fifth process is to try to extract the dioxin from the soil—some sort of solvent-extraction process. Frankly, the state of the art just is not there; I do not expect it to be there, although I am not a scientist, for some period of time. And I am being advised by my technical people that if and when that emerges as a solution, the remedy would take approximately 11 years, the cost would be $58 to $77 million, based on current estimates, and the extracted dioxin from the soil probably would produce a byproduct that is more toxic than the dioxin.

Mr. LEVITAS. Thank you, Mr. Hedeman.

Thank you, Ms. Johnson. Your time has expired, and we will get back to you.

Mr. Wise.

Mr. WISE. Dr. Hernandez, returning to Dow again, you stated that you directed that the report be given to Dow Chemical Co. Did you at the same time direct that it be given to any consumer groups, the Sierra Club, the League of Conservation Voters, and citizens' groups in Mr. Albosta's district, for instance?

Dr. HERNANDEZ. I think I have indicated that I did not suggest distributing it to anyone else, other than to them and to the State of Michigan.

Mr. WISE. Would they not also be similarly affected, as Dow is affected?

Dr. HERNANDEZ. You know, I believe that the EPA has many competent people. All of us believe that we are environmentalists who view our concern or that of public health and the protection of people, and that seeking Dow's input on this was not inappropriate.

Mr. WISE. But if you sought Dow's input, would it not have been appropriate to seek others' input who do not always share the confidence that EPA is an environmental group?

Dr. HERNANDEZ. As I said, Mr. Wise, one mistake I probably made was not sending it to a large number of people so we could have overcome that criticism, and I think that is a valid criticism.

Mr. WISE. Do you agree with the remark attributed to the region V administrator, Val Adamkus, that the action was highly improper?

Dr. HERNANDEZ. You know, I do not know what the regional administrator did say. It is a quote. I think that to make that kind of judgment call 18 months down the line, when he was working for Anne Gorsuch just the same as I was, and he had an opportunity to suggest to her that it was improper, and we certainly would have honored that or looked at it. He never raised the issue with me before——

Mr. WISE. Did you ask him before you directed that the report be released?

Dr. HERNANDEZ. I had a conversation with him—and I was the one who released the report to Dow and to the State of Michigan, too——

Mr. WISE. You had a conversation. Did he object at that time?

Dr. HERNANDEZ. Not that I recall.

Mr. WISE. So to your knowledge, there was no objection at that time?

Dr. Hernandez. Not one that was voiced in any way that said: "Gee, I think that is a really bad idea."

Mr. Wise. Well, was there any kind of objection, perhaps implicit? I know that I would probably have some reservation in telling a Deputy Administrator that it is a really bad idea.

Dr. Hernandez. I do not feel that is the case. I think people who have gotten to know me know that I love the give and take of arguments, and I love to debate these issues—as I have kind of indicated today.

Mr. Wise. Well, did you check with him—after you directed the report—did you have any conversations concerning this?

Dr. Hernandez. I think it was a very short one, and it may have been in the context of something else, but not—I think I talked to him twice on the issue.

Mr. Wise. Are there other situations where you directed that a report be given to a company directly affected?

Dr. Hernandez. I cannot think of one, but there could have been, there could have been. You know, we deal with so many issues like this, and trying to decide that in some context, you have not, it is pretty hard to say absolutely no. I just cannot recall one at this time.

Mr. Wise. It stated in the Washington Post article that part of what was deleted was a recommendation that fish consumption from the two Michigan rivers be prohibited, but an EPA spokesman said officials in Washington decided this was an FDA matter. Was the FDA then informed that there was a possibility of contamination in these rivers, and that they should look into it?

Dr. Hernandez. I have no idea, Mr. Wise, because I did not know what was deleted, what was retained, whether or not we did discuss in headquarters that that was an FDA level and that the decisions would be based on their level, and EPA does not set levels for contaminants in food, and for us to make that kind of a statement in a report, I believe they would become a little bit concerned about us moving into their territory. That is a judicial issue.

Mr. Albosta. Would the gentleman yield?

Mr. Wise. Yes.

Mr. Albosta. Do you mean to tell me that you and the Food and Drug Administration do not get along, and you cannot exchange information?

Dr. Hernandez. No, sir, I did not say that, sir.

Mr. Albosta. Well, when you determine that something is as toxic as dioxin is, do you mean that you do not tell the Federal Food and Drug Administration what the results are? That is what I am asking.

Dr. Hernandez. No, sir, we——

Mr. Albosta. I cannot believe you. You just said that, though.

Dr. Hernandez. No, I did not say that, sir.

Mr. Albosta. Dr. Hernandez, what did you say?

Dr. Hernandez. I said that for us to come to a conclusion——

Mr. Albosta. You said that that would be rivalry.

Mr. Hernandez. No, sir. Could I try again?

Mr. Albosta. Sure, go ahead.

Mr. Hernandez. For us to make a conclusion in a report that it might be dangerous to eat any fish in the Great Lakes would be a judgment decision in a process that was not typically within our mandate. Ours has to do with exposure on emissions from air, or water consumption, where we have standards. Those are FDA standards in this issue.

Mr. **Albosta**. But just a minute. Wouldn!t you say to the FDA that, "From our studies, this is a very toxic material in water, and we would assume that if it is in fish, it is just as toxic"? Do you mean you would not say anything like that? That is exactly how I took your answer.

Dr. **Hernandez**. Well, we do work with FDA, and I have with us today Dr. Barnes, who is head of this Chlorinated Dioxin Work Group that deals with FDA on those levels, and I think it is appropriate to have him tell you what kind of relationship we have with them, if that is appropriate.

Could we ask him to be sworn in and answer that specific question about FDA?

Mr. **Wise**. But what I am concerned about is, regardless of the relationship that you share, there is apparently a finding that you have a very toxic substance in the water. There is a toxic substance in the fish. You delete it from the report, and I want to know whether FDA ever found out about it.

Dr. **Hernandez**. Could I ask Dr. Barnes to answer that?

Mr. **Levitas**. If you could first answer the question, do you know whether FDA found out.

Dr. **Hernandez**. And I said no to that answer; I do not know, I do not know.

Mr. **Levitas**. Mr. Wise, would you like to hear a response from Dr. Barnes?

Mr. **Wise**. Yes, but before I do I have one other question that concerns me, and then I would be glad to hear, if I have any time remaining, from Dr. Barnes.

You stated earlier that in terms of the report, it was not connected with any enforcement action, it was not connected with any regulatory action. It was simply a scientific report, and therefore you did not feel the same constraints on releasing that to Dow that you might have on a more sensitive matter—at least, that is the way I took it. Is that accurate?

Dr. **Hernandez**. I think that is correct. To my knowledge, this was not written for any kind of an enforcement action. There was nothing in it to indicate that it was an enforcement paper. It was a set of informations on dioxins over a wide range of things, everywhere from waste cumbusters to automobiles to powerplants, and it was designed to give an overall picture of dioxin emissions within the Great Lakes area.

Mr. **Wise**. Yes, sir, but because of some of what turned up, it could then be used in an enforcement action or in a regulatory action, could it not?

Dr. **Hernandez**. Certainly, all of our technical information is what underpins those things.

Mr. **Wise**. And finally, before we hear from Mr. Barnes, if that is the chairman's will, I guess I would like to say that we spent several hours here, just debating what was and was not in that report and what was deleted. What concerns me is that we may have lost track of, regardless of what was in the report, what is out there;

what dioxins are there; what dangers exist. I mean, we take out the references to toxic carcinogens. Well, I understand that Dow may not want that in the report, or that this may be an element of agent orange-related problems and that Dow may not want that in the report, but the fact is, what is out there. I think we have gotten too much concerned about what the final report said, and we have not concerned ourselves with whether there is indeed a danger there and the people ought to know that.

Dr. HERNANDEZ. As I said, the report has gone to the State of Michigan and was available to them, very quickly after the draft report was made available.

Mr. WISE. Thank you.

Mr. LEVITAS. Dr. Barnes, I am going to swear you in, and then if you could briefly respond to the question.

Do you solemnly swear that the testimony you will give before this committee will be the truth, the whole truth, and nothing but the truth, so help you, God?

Dr. BARNES. I do.

Mr. LEVITAS. Please give your name and position, and then if you could respond briefly to the question.

TESTIMONY OF DR. DONALD BARNES, SCIENCE ADVISER TO THE ASSISTANT ADMINISTRATOR FOR PESTICIDES AND TOXIC SUBSTANCES

Dr. BARNES. I am Dr. Donald Barnes, science advisor to the Assistant Administrator for Pesticides and Toxic Substances.

The question, as I recall, is the relationship between EPA and FDA vis-a-vis the question of dioxin contamination in the fish.

At the time that this report came to the Agency in May 1981, we were in the midst of working with FDA on an international exchange between the United States and Canada, concerning the question of dioxin contamination in the Great Lakes. We had had a meeting at the State Department, I believe, in December 1980, in which a delegation had come down from Canada to lay before us data that indicated that there was possible dioxin contamination in the wildlife, particularly in that case, birds, in the Great Lakes area. We had already in hand some data that appears in this report, as a matter of fact. There were data provided by the U.S. Fish and Wildlife Service and others, all of which was presented at that meeting. As I recall, the FDA was fully cognizant of it at that time. At that December meeting, the decision was made that as a Federal Government, the FDA would take the lead on this question vis-a-vis dioxin in the fish in relationship with Canada.

This report from the region came in, as I say, in the spring of 1981. We had had scheduled, as I recall, at that time, a meeting in Ottawa, Canada, to discuss the results of the December meeting. At the December meeting we decided, "Look, we need to get some more data. We need to go out and get some more fish samples. We will meet back in Ottawa in 6 months."

So, during that 6-month interval, we were working with FDA on a continuing basis regarding the specific question that you have addressed.

Mr. WISE. Then, the reference that was in the original report, that fish consumption in the two Michigan rivers be prohibited, is it now both FDA's position and EPA's that that is not necessary?

Dr. BARNES. FDA as a result of our meetings in Ottawa, Canada, issued a letter to each of the Governors of States bordering on the Great Lakes, in which they advised—because the question was there were so many sports fisheries over which FDA does not have direct concern—but they offered the advice that levels of less than 25 parts per trillion in fish would not be a problem; if it is between 25 and 50 parts per trillion, then you should limit your consumption, and if it is greater than 50 parts per trillion, then you ought to look clearly at whether or not to eat that fish.

Mr. WISE. But when I buy a fish, I do not measure the parts in there. Should people on those two rivers be eating those fish or not? That is the question.

Dr. BARNES. Well, my understanding was that that was the advice that was given to the Governors of the States, to allow them to make the decision. And I am not sure, but I believe that the Governor of Michigan did act on that vis-a-vis the Tittabawassee River.

Mr. WISE. Mr. Chairman, I still do not know. I still do not know whether you should eat the fish in that river or not.

Mr. LEVITAS. Do you know, Dr. Barnes, whether fish in that river with those levels of dioxin should be eaten? Do you have an opinion on that?

Dr. BARNES. To say what is going to be the outcome or the consequences of that——

Mr. LEVITAS. No. I just said do you have any opinion on that.

Dr. BARNES. Do I have any opinion? My personal opinion is that if I were asked to eat fish that I knew had 20 parts per trillion of TCDD in it, I would not feel as though that would be a problem.

Mr. SNYDER. Would the gentleman yield to me?

Mr. WISE. Yes.

Mr. SNYDER. Along this same line, Dr. Barnes, you had brought up the meetings that you were having with Canada about the time of the "Dow problem." Were not there other studies involving toxics in particular, not limited, though, to dioxins, but all sorts of toxic chemicals, going on under the auspices of the International Joint Commission?

Dr. BARNES. I believe there were. I do not know. That group was included in the loop at the State Department. They were heavily represented, and I think it was under some of their auspices that some of these samples were collected during that 6-month period.

Mr. SNYDER. And weren't there other, similar studies being conducted by various States in regard to their own internal problems?

Dr. BARNES. Yes. New York State was another participant that came up there, and they had been looking specifically at issues in New York, and they analyzed fish as well.

Mr. SNYDER. Now, what is your input or what do you receive as a result of these other studies I do not know whether you have input into the process or whether you simply get information from these studies. Can you tell us how the process works?

Dr. BARNES. Well, those data come in to us. As I perceive the sequence, there are three questions. One is whether or not you ought to eat the fish, and that is something that the people in the FDA analyzed. Where the EPA comes in is the question of where does it come from and what are you going to do about it.

Mr. SNYDER. Is there any substantial inconsistency, so far as you have been able to determine, between these various State studies and/or the International Joint Commission study and the Dow study, if we may refer to it as the Dow study?

Dr. BARNES. Not in terms of what the numbers are. I mean, everybody seems to agree that—in the survey of fish in the Great Lakes, as I recall, there were something like maybe 200 samples taken——

Mr. SNYDER. So if there is any complaint about this study, it is not about the result?

Dr. BARNES. Not in terms of the numbers.

Mr. SNYDER. The result in the final Dow draft, so far as you know, is consistent with the results that have been obtained in similar studies in other places.

Dr. BARNES. Well, certainly, as far as the analytical results are concerned, that is the case. I have not read the other ones, so I do not know what their conclusions and summaries are.

Mr. SNYDER. I thank the chairman.

Mr. LEVITAS. Thank you, Mr. Snyder.

Before I get back to Dr. Hernandez, Dr. Barnes, you say you are science advisor to the Deputy Administrator?

Dr. BARNES. No, to the Assistant Administrator.

Mr. LEVITAS. The Assistant Administrator. And that is Mr. Todd Hunter?

Dr. BARNES. Yes.

Mr. LEVITAS. Are you an employee of EPA or a consultant?

Dr. BARNES. I am an employee.

Mr. LEVITAS. How long have you been employed with EPA?

Dr. BARNES. Three and a half years.

Mr. LEVITAS. And what did you do before coming to EPA?

Dr. BARNES. I taught at a college for 12 years.

Mr. LEVITAS. And where was that?

Dr. BARNES. At St. Andrew's Presbyterian College in Laurinburg, NC.

Mr. LEVITAS. Thank you.

Dr. Hernandez, I have a series of questions I want to try to put to you to try to deal with, and, perhaps, dispose of an entire area. But one of the things that has come to my thinking, and I just want to make this observation, after listening to weeks of testimony about problems at EPA and the Superfund, I really get the feeling that the words, "We have made a bad mistake," or "We have had a bad policy," or "We have been wrong," are just missing from our political lexicon. And if people in the Government—and that is true of Congress, as well as in administrations and the office of the President—had simply said years ago, "We were wrong"—if President Nixon had said, "I was wrong"; if President Ford had said, "I should not have pardoned him"; if President Carter had said he had made a mistake; if President Reagan said, "I have embarked on the wrong policy, and I want to change it"—I think a lot of our problems would go away. If we could just get the "mea culpa" back into our lexicon, I think a lot of people in the public and in Congress would begin to work constructively to get on with the problem from this point forward.

Dr. HERNANDEZ. I think that is a good observation.

Mr. LEVITAS. Let me put in context some of the concerns that people are having about EPA, as I see it, and certainly in this committee.

There is a feeling that EPA in the last few years has had too close a relationship with industry—I am not saying these are true, but let the facts speak for themselves—there has been too close a relationship—whether it was Ms. Lavelle's gastronomical experiences in Washington, or whether it was your decision to release this report to Dow. But there is that feeling, and I want to explore some of that.

Another concern we have is that it has taken too long to get on with the problems of cleanup; that there have been unconscionable delays before action has been taken.

Now, I mentioned the submission of this report to Dow, this dioxin report. Let me give you another example. You were involved, as I recall, in the matter of deciding what to do about formaldehyde regulations, and I believe one of the places you turned to get information about how to deal with that was the Formaldehyde Institute; is that correct?

Dr. HERNANDEZ. They asked us for an opportunity to meet with our staff. You know, I have caught some flack on that one, both in the scientific community and in other places, and in all candor, I would not do that again, because I think there is a better way to do it. Coming out of academia, and when you find people saying, I believe this—and I am talking about scientific information—we take the same kind of data set, and "I believe this is the interpretation," and these other people say, "Here is how I interpret that scientific data," the way you resolve some of those is you get people together and you say, "Let's talk about it." That is what those were.

Mr. LEVITAS. But you understand what the perception is, that the Formaldehyde Institute had a specific and vested interest in this matter. Whether it was a good decision or a bad decision, I am not passing judgment on at this time. The Dow situation is another example, and we have addressed that pretty extensively.

Let me give you another situation that creates this perception. It has been called to my attention that there was a situation that occurred in early 1981, specifically, a discovery by EPA officials of exceptionally high levels of lead contamination in areas surrounding three lead smelting operations in Dallas, TX, involving the National Lead Industries, Dixie Metals, and RSR Corp.'s smelting operations. Specifically, over the last 2 years, EPA and other test data have, according to some of the reports, furnished the following information—that soil texts showed lead concentrations of 1,000 to 3,000 parts per million in playgrounds of several of the local schools in the area and in the homes, as well; in some areas, children might actually be exposed to levels up to 10,000 parts per million, and at one location, the West Dallas Boys' Club, at almost 20,000 parts per million, a level that is 60 times the level considered safe by medical researchers. In homes near two of the smelters, homes contained even higher levels, up to 25,000 parts per million; that as many as 70 percent of the residents who live in the areas immediately adjacent or near these three lead smelters have extremely high levels of this toxic metal in their systems, and that more than a dozen children in this predominantly black, poor subdivision in the Dallas area may have life-threatening levels of lead in their blood; and that a recently released study done by the Center for Disease Control in Atlanta, funded by EPA, shows that while overall test results of the lead blood levels were below the national average, they were 14 percent higher than the norm for the Dallas area.

Now, I have mentioned all of this because, first of all, EPA has known that these problems have existed for slightly more than 2 years, and as I understand it, virtually nothing has been done to clean up the lead soil deposits, and only now are the EPA and the State moving to control air emissions from one of the two smelters and to eliminate part of the problem. And according to news accounts, you, Dr. Hernandez, more than 18 months ago, were quoted as saying, "We are proposing some approaches to the lead contamination issue that will be scientifically sound and that will be within the context of practicality of our budgetary restraints." Lead is a very hazardous chemical. It can damage the central nervous system, kidney damage, is known to lower the IQ of children and impede their mental accuity. All this was known 18 months ago, but nothing was done. Why?

Dr. **Hernandez**. I think a great deal was done on that particular one. First, there was a need to characterize the problem. What we started off with at that time was a lead-in-soil study. We did not have good ambient backgrounds around those areas for the air emissions to tie with that, and we did not have the kind of information needed with respect as to exposure, to particularly children, but also individuals in that area as a whole. What we really needed was a sampling program where we went out and actually measured people——

Mr. **Levitas**. A sampling of what?

Dr. **Hernandez**. Blood levels in children, particularly.

Mr. **Levitas**. Why would you need to have that information if you knew what the levels of lead were?

Dr. **Hernandez**. There is no a priori lead level in soils that is a prerequisite for some kind of cleanup action. The route of soil lead into the body system, the exposure system, is complex, depending on the age of the children, the kind of climate you have, dirt streets versus paved, and as a result we do not have an a priori cleanup level. There is nothing that says, "Here is the place at which you begin to remove soil automatically."

There were some initial things that happened in that case——

Mr. **Levitas**. But before we get away from that, you remember I asked you earlier about reliance on epidemiological evidence as opposed to human test evidence? I am not a scientist, but I have read for years, going back as far as the Romans, that people knew that exposure to lead and high quantities of lead had these consequences. High levels of lead were determined to be in place at these three sites in Dallas. Why did we need to take blood levels of these children for 2 years before something was done about cleaning up the mess?

Dr. HERNANDEZ. Well, those lead levels which we found in the initial EPA soil survey—that was an EPA study that came out sometime in mid-1981—indicated kind of a plume pattern, if you can picture it, going away from these places with higher soil lead levels closer to the plants. Some of those plants had been shut down, and others were still operating. As I said, there is no a priori cleanup level for lead. There was some initial action by one of the companies to clean up a playground that was very close to their property—take some of the soil away—but the rest of it, what we wanted to do was try to characterize the problem in terms of people; what was that exposure route—because when you look at lead, it is contributed in three principal routes—one, the food you eat every day; two, the dust that you are exposed to both within your home and the surrounding yards and things like that, and three, through the air. And some of the lead data that we have in air in urban areas, compared to blood lead levels in youngsters shows, that as we go into leadfree gasoline, there is a comparable decrease in lead in blood levels in children. We had to have that kind of data in order to base any kind of remedial action.

Mr. LEVITAS. But I am not really sure you did, and I think that is part of my problem. We know that lead has a cumulative effect. It comes from paint, it comes from the air, it comes from the sources you have described. Now, I understand that in region VI, the Dallas region, a toxic substance program toxologist wrote a memorandum to the region's enforcement staff in the fall of 1981, telling them that there was or is a serious problem that needs to be resolved, and that the enforcement staff was working out an arrangement whereby the three smelting companies that were involved--not the government, not the taxpayers, but three companies—would foot the bill to clean up the lead-contaminated soil. And this was going to be done right away. This gets back to my problem of why it takes so long to clean up something and expose people over those periods of time. It would have been done right away, not 2 years later, after a lot of blood had been pulled from a lot of kids.

Now, these three companies were going to clean up the area, but they were precluded from doing so because someone at headquarters of EPA refused to allow the regional enforcement staff to go ahead and conclude this settlement and permit them to do so. Now, are you aware of that?

Dr. HERNANDEZ. Well, I followed that pretty carefully, and I do not recall that there was ever a statement that said, "You may not move ahead with enforcement." I just do not recall that at all, that kind of characterization. I think my involvement was to get ERA to design a good, stratified sampling process that would allow us to say, "There is lead in the soils out there. It appears to be related directly to the exposure levels in these youngsters in this area, and here is where we ought to do this kind of cleanup process.

An immediate blood lead sampling program went into effect. Unfortunately, it was not very well-stratified, and people came from all over town. Eventually, we did get a good blood-lead sample, the one that you referred to finally took place.

But in terms of precluding some kind of enforcement, I just do not believe that we ever said anything like that.

Mr. LEVITAS. I did not say "preclude enforcement." That is another subject altogether.

First of all—let me go back—did you know about the toxicologist's memorandum to the enforcement staff that there was or is a serious problem that needed to be resolved?

Dr. HERNANDEZ. No, I did not.

Mr. LEVITAS. Did you know that there were negotiations which had been undertaken with these smelting companies to undertake a cleanup?

Dr. HERNANDEZ. Yes.

Mr. LEVITAS. And did you know that the companies had agreed to do a cleanup?

Dr. HERNANDEZ. I never really knew that they had agreed to do any specific kinds of clean up. There was some talk of doing this or doing that, and I think one of the companies was quite willing to do something. I am not sure about the others.

Mr. LEVITAS. Did you tell them not to do it?

Dr. HERNANDEZ. I did not tell them not to do it.

Mr. LEVITAS. Did you tell them to go forward and work out that agreement, so they could get on with it?

Dr. HERNANDEZ. I do not think it was ever presented in that kind of a context, where there were direct choices of, "Let's go and get the cleanup," versus, "Let's do the scientific study." I do not think that was ever any kind of a choice that had to be made.

Mr. LEVITAS. You see, Dr. Hernandez, what bothers me about this—and it gets back to several other things, why it takes so long to get something done—here, we had evidence of high lead concentrations in the soil, and in the environment around these three smelting plants. EPA's toxicologist, according to our information, said there was a serious problem. The regional enforcement people were saying, "We will negotiate with these smelting companies," and I am advised that they had agreed to do the cleanup. When it got to headquarters, I am advised that someone in headquarters EPA said, "Hold on. We are going to do blood tests on these children to find out what the level of their blood lead is before we do anything," instead of saying, "Let's clean it up."

Now, did you know about that—or, Mr. Daniel. Is Mr. Daniel still here? Would you know about that, Mr. Daniel?

Dr. HERNANDEZ. I do not think he would know——

Mr. LEVITAS. Did you know about that, Mr. Daniel?

Mr. DANIEL. I did not.

Dr. HERNANDEZ. I think I was the one who was basically following that.

Mr. LEVITAS. It was my understanding that you were the person.

Dr. HERNANDEZ. Yes, I think that is correct.

Mr. LEVITAS. But why should we expose children to high levels of these chemicals—lead, in this instance—while we are taking blood samples, instead of cleaning it up, when the companies there were prepared to go forward and do so?

Dr. HERNANDEZ. Let me first say that there are a number of places in the United States that have significantly higher than average lead soils around the United States, where we have not taken any outright action. What we have done in each of these cases is to look at the relationship between those lead soils and the lead levels in the children in that area and to determine whether or not there was a serious problem, and where there has been a serious problem, we have recommended some kind of recourse. And I believe that is the appropriate process.

Mr. LEVITAS. You think it is appropriate where there are identified high levels of lead in an area, identified by EPA's own scientists, where the smelters involved had indicated a willingness to do something, to hold that up and test the blood levels of these children and let them be exposed continuously through that period?

Dr. HERNANDEZ. Well, let me say two things again here.

Mr. LEVITAS. I so fundamentally disagree with that, Dr. Hernandez. And I am not trying to substitute my scientific judgment for anybody else's. I am simply saying where you have a contaminated situation, and somebody is willing to go forward, why should EPA be the one to say, "Hey, hold off, don't do it?"

Dr. HERNANDEZ. Let me say the degree to which they wanted to go forward, the readiness to go forward, I do not have any good notion about that.

Mr. LEVITAS. Well, you were not looking at it, Dr. Hernandez. You were the person at headquarters, you said, who was sort of monitoring this.

Dr. HERNANDEZ. Well, you know, we have a Regional Administrator. If he felt that was the right course of action, we would have considered that and followed a recommendation. We are in partnership with a number of other people in every one of these cases—the Dallas Health Department, the Texas State Health Department, the Center for Communicable Disease Control, and EPA, in making this kind of an evaluation and decisions on it.

By and large, we felt the best process in this thing was to go forward and find out the kinds of scientific information as to the exposures that we were having. Now, when we got that information, we found that the percentage of children that exceeded a target level identified as a concern level was not significantly different from that in large cities in the United States, even though it was somewhat above that, in terms of percentages, that you have for the remainder of Dallas.

But remember this, that any time you have an average figure, you have some people above and some people below.

Mr. LEVITAS. Dr. Hernandez, what I am suggesting is something very fundamental. I am suggesting that where there are high levels of lead in the environment, unusually high levels of lead in the environment, and there is a willingness on the part of the people who were responsible for putting that lead there, why not go forward and clean it up? Why use the children as scientific test animals for purposes of finding out just what is wrong, before you clean the problem up? That is what I want to know.

Mr. Daniel, did you know about this situation? Are you involved in this decision?

Mr. DANIEL. Mr. Chairman, I knew that the sampling of blood levels was going on in children. I have never heard that there was any negotiations with any of the companies there, or that there was any offer to do anything.

Mr. LEVITAS. You were not aware of that?

Mr. DANIEL. I was not aware of that.

Mr. LEVITAS. All right.

Dr. HERNANDEZ. Mr. Chairman, let me just say the characterization of using the children as guinea pigs is just not fair, and that is not characteristic of what we were doing.

Mr. LEVITAS. I am not suggesting that.

Dr. HERNANDEZ. We were using the best information we had from this team of people looking at the situation.

Mr. LEVITAS. Who made the decision to take the blood tests rather than do the cleanup?

Dr. HERNANDEZ. Well, I do not think there was ever an either/or decision. I think that there was a consistent willingness of one of the companies to do something. Now, whether or not that was total and appropriate or not, the only way you could determine it was by finishing the study process.

Now, I would say you would have a really good point that you could say, "Hernandez, look at these blood levels in these kids. You should have taken action." I do not believe that was the outcome.

Mr. LEVITAS. Dr. Hernandez, you have put your finger on where you and I disagree about this thing. I am suggesting where the Environmental Protection Agency identifies the presence of a potential health hazard in the environment, that the thing to do first is to clean it up, to eliminate the risk.

Is there anybody——

Dr. HERNANDEZ. And I was saying, Mr. Chairman, that there are no magic numbers that you can look for—criteria for lead in soils to make that kind of a decision.

Mr. LEVITAS. Let me make sure I understand your testimony quite clearly. You were unaware that the companies involved, the smelters involved, had negotiated with the regional enforcement people for purposes of cleaning up the soil, removing the soil?

Dr. HERNANDEZ. I was familiar with the fact that at least one of the companies would do something, but the size of that plume, where it was, and how much cleanup was necessary, and under what kinds of situations, was not clear at all. You know, we may have made one of those—let me just give you the other side. Supposing that we had, the first day that report turned up, said, "Let's go and take that playground and clean that place up, and they do this, and say 'Done' and get a settlement out of it," and then we would have been charged with a "sweetheart" deal with somebody, done in the dark of night, before we had done any kind of further study, when in fact, we found that this study showed problems further out.

Mr. LEVITAS. But if you had let them clean it up and not released them from further liability, if they were willing to clean it up, you would have eliminated 18 months of exposure.

Dr. HERNANDEZ. And I do not know that they were ready to take that kind of thing, to do some kind of cleanup and also remain liable. I do not know that.

And listen—I was never informed about that kind of detail on this thing, as to where we were at in that kind of enforcement process.

Mr. LEVITAS. You see, again, it gets back to—let us relate it strictly back to the Superfund situation. One of the concerns that we on this subcommittee have in the philosophy that negotiate first and litigate second has had is it has overlooked completely the purpose of Superfund. You did not need Superfund to negotiate; you did not need Superfund to litigate. Those were there to begin with. Superfund had as its purpose to clean up, clean up the sites, remove the health hazard, and take people out of jeopardy.

Now, here, in the Dallas lead matter, I see a situation—and the reason I am pressing you on this so is that I want to see if we can bring about a change in attitude and philosophy, not just a change in personnel, but a change in attitude and philosophy, which says where you identify the potential of contamination that could potentially pose an imminent health hazard to people, get on with the cleanup and do the scientific blood sampling later on to see how far you have to go, but not to wait 18 months before you do the initial action—particularly in the Dallas situation where, according to the information I have—and I suggest you go back and have someone review the files at headquarters—there was a willingness on the part of the smelters to go forward through a negotiated settlement at their expense.

Now you can respond.

Dr. **Hernandez.** Let me say that in terms of emergency situations where there is a clear-cut real threat to humans, we do move ahead' we do clean it up. We do not wait. We step forward and do it.

Lead soils, where you do not have an a priori target that says, "This is an emergency," at least at the levels we have been dealing with, "This is an emergency. Move forward with great haste here." You know, if you said, "I want to change your mind about using good information, about collecting the best information, about looking at exposures," you know, risks to human health are based on two things—one, the presence of a toxic material whose risk we analyze; and the second one is the exposure and the exposure route that you have. And it is through those two processes, neither one independent of the other, that we make our decisions.

And you know, I want to be responsive to you. I want to say, "Yes, we are going to move out there and move forcefully."

Mr. **Levitas.** I understand, and I respect your position on this, Dr. Hernandez. I am just telling you we have a difference of approach in how we ought to deal with this, and I want to explain in a moment how EPA in another circumstance reversed its priorities of timeliness. But please, I want you to finish. I want you to state your position for the record.

Dr. **Hernandez.** I guess, whether in the absence of any kind of clear-cut criteria for target levels for concern for the presence of a particular chemical in the atmosphere, in our water, that some continuation even for a short period of time would be hazardous—moving ahead without some kind of information as to the nature of the source, the mode of travel, the kinds of control techniques that might otherwise be used, would leave us open to the same kinds of criticisms, "Why did you do that?"

What I am saying to you is that on every one of these cases, we just have to use our best judgment and use our best guess on these things.

Mr. **Levitas.** I would like for you, Dr. Hernandez, to review the files on this matter. I am asking our staff to make arrangements to go through these files, just to establish the chronology of events that I have described.

But let me give you the other side of the coin. You see, I think that when there is the potential of a health exposure, you ought to move quickly, and then if you are going to err, err on the side of prevention rather than on the side of delay.

About the same time this was going on, you will remember the matter of the Thriftway Co., the New Mexico refinery. In that situation, according to what has been discussed publicly, Mrs. Gorsuch, as administrator, had made a commitment to this small refinery that they could exceed the standards—I think it was lead involved in that case, as well—while EPA was reconsidering those standards in the regulatory effort. Now, what happened in that case, chronologically, was that EPA through its administrator said, "Look, you have a problem right now. You take care of your problem. Do not worry. We are not going to enforce it against you, because we are in the process of doing something about those things dealing with lead." Here, you have a situation where a community has found high levels of lead; there is the possibility of an immediate problem or one that could certainly be aggravated by delay, and the philosophy was, "Let us hold off until we get all the facts in, before we make a change in our policy." And what I am saying is that the community in Dallas at least should have had the same priority consideration given to avoiding a health problem as the Thriftway Co. had in being relieved of the economic burden that was being imposed upon it.

It is a philosophy of how you deal with these matters that goes back to the concern that I have of the public perception of the Agency now. And it is that which I want to clean away, so this Agency can get back to establish its credibility and do the job that the people at EPA should be doing, and I know that many of them want to do.

Dr. HERNANDEZ. We share that. We share that same basic concern, and we need to do it, because this very establishment called EPA will not regain its health overnight. You all have to help in that process.

Mr. LEVITAS. Well, that is what we are trying to do, Dr. Hernandez.

Mr. ALBOSTA. Would the chairman yield on that same subject?

Mr. LEVITAS. I will be happy to yield on that subject, and then I am going to recognize the gentleman from New York.

Mr. ALBOSTA. Dr. Hernandez, what would your opinion be of Times Beach, MO?

Dr. HERNANDEZ. You know, I really do not know that much about the last set of data of information collected. Mr. Hedeman knows much more and knows the details as he participated in that issue.

Mr. ALBOSTA. But you expressed yourself that you ought to do all this testing, and you ought to go on for years and test people, and so on.

Dr. HERNANDEZ. No. Sorry. Please, do not say that kind of thing. What I said about the Dallas lead problem was that it was a short-term testing program. We had a finite group of people, a well-run testing program; it had a beginning and it had an end. We are not talking about leaving people to be exposed without some kind of understanding of the exposure routes. Lead is different in its impact, and our ability to notice it in young children. You can notice it very, very quickly.

Please, do not take the kind of answer—what you are trying to do is to take and generalize it into some kind of philosophical statement, on the basis of what I am telling you. Let us collect good information. Let us do it in a timely fashion. When it is necessary, let us act, and let us go on and correct the problem. I do not have that problem at all.

Mr. ALBOSTA. Well, I am pleased to hear that, and I hope in Midland, MI, that that is the case, also, that you will take some action, and you will act.

Dr. HERNANDEZ. We continue to do monitoring out there, and we will have another report out quickly.

Mr. LEVITAS. Thank you. Just one last question, and then I have got to ask Mr. McPhee a question.

I want to get back to my earlier question about relying upon general scientific information concerning the effect of certain substances, reliance upon environmental tests and studies, general scientific literature, epidemiological information—and as I say in the case of lead, it goes back to the Romans—as opposed to having to rely upon human studies as the basis for regulatory or decisionmaking policy.

Dr. HERNANDEZ. Epidemiology is basically human data.

Mr. LEVITAS. Yes, but it is historical, and it is broad, and it is based upon the compilation of experience without a particular directed test.

Do you feel that the reliance upon this general body of scientific information, and animal studies, can serve as a basis for regulatory action by EPA, or do you feel it has to be based upon human studies?

Dr. HERNANDEZ. Oh, no. Basically, on some organic chemicals, where we have little other information other than animal testing, that is what we have to use. What we try to do, and what I hope we do is to use as large a base of scientific information on any one chemical in the decisionmaking process.

Mr. LEVITAS. Thank you.

Mr. McPhee, I want to get something clarified. You stated that the litigation with Dow was commenced on December 15 of last year, but that discussions and requests for information had been extending over a period of time. To the best of your recollection, can you identify the time at which the first request was made of Dow Chemical for the information you were seeking with respect to the dioxin?

Mr. MCPHEE. Well, dioxin was one of the many substances that apparently are discharged from the Dow plant. The initial request covered that whole, broad spectrum, and I believe it was originally laid on Dow on January 21, 1981.

Mr. LEVITAS. And it was in December, 11 months later, that the litigation was commenced——

Mr. MCPHEE. I am sorry. It was 1980. It was approximately 2 years going on.

Mr. LEVITAS. Two years.

Do you see the problem, Dr. Hernandez?

Dr. **Hernandez**. And I can see this comes back to that same one—do you move ahead, or do you try to litigate. And clearly, the region, in good faith, tried to negotiate, and probably did get some information. I do not know what they received from Dow. But I think that in good faith, you try that occasionally.

One of the questions asked by Mr. Andrews bears on this. I said that we move forward to first find information, and then we make some decisions. In criminal cases, we should move like that. There is no negotiation in criminal cases.

Mr. **Levitas**. There have not been any criminal cases in Superfund, except for one spill case that I am aware of.

Dr. **Hernandez**. There can be down the line. I am talking in the general sense. With RCRA, there are going to be lots of opportunities, and we should move on those.

Mr. **Levitas**. Thank you. I have taken much too long on that line.

The gentleman from New York.

[Whereupon, Mr. Andrews assumed the chair.]

Mr. **Molinari**. Dr. Hernandez, isn't it a fact that in region V, there were some accusations made by employees of EPA and headquarters that region V was moving ahead too fast, without getting the approval from headquarters of EPA?

Dr. **Hernandez**. On which issue?

Mr. **Molinari**. Well, one, for example, is the Berlin and Farro site.

Dr. **Hernandez**. I really do not know much about those. I would have to ask Mr. Hedeman to talk to you about that. I am not familiar with those.

Mr. **Molinari**. Well, Mr. Hedeman, I think we discussed this in the past. I am just picking up where the chairman left off. He raised the question in several instances that they were not moving quickly enough. Yet, in questioning witnesses, I have found a great deal of criticism levelled at region V with regard to negotiating and moving ahead with settlement too quickly without getting the approval of EPA in Washington.

Mr. Hedeman, is that not a fact?

Mr. **Hedeman**. When you raised the question about Berlin and Farro, I was somewhat taken by surprise——

Mr. **Molinari**. Mr. Daniel, do you know anything about that? You were chief of staff.

Mr. **Daniel**. The only thing I know about Berlin and Farro is that Mr. Dingell has subpenaed documents relating to those sites. I know nothing about any of the enforcement actions relative to Berlin and Farro sites at all, either action, inaction, or otherwise.

Mr. **Molinari**. Well, is there anybody here from the EPA who can tell me whether I am wrong in what I have said?

Mr. Lucero.

Mr. **Lucero**. Mr. Molinari, as I recall, the issue there—or, at least, the issue I am famliar with—was that the region had wanted to move very quickly on some activities——

Mr. **Albosta**. Mr. Chairman, point of order. Has this witness been sworn in?

Mr. **Andrews**. Yes; he has been.

Mr. ALBOSTA. Thank you.

Mr. LUCERO [continuing]. That the region wanted to move ahead and about certain activities under an emergency that questions were raised as to whether they were not indeed a remedial action for which a State share might be required.

We saw a lot of discussion over that issue. I think generally, you are right. The region did want to move quickly at the site. They felt considerable pressure because of public health threats and others to do that, and there were some policies, namely, how the activity would be characterized and whether it would require a State's share, and some questions about giving notice to the responsible parties before the work was done that they ran up against.

Mr. MOLINARI. Isn't it fair to say there was some criticism from headquarters as to the manner in which they were moving, perhaps too quickly, without consulting with headquarters?

Mr. LUCERO. I think that is right.

Mr. MOLINARI. Thank you, Mr. Lucero.

Dr. Hernandez, who is P. Edes Rosius?

Dr. HERNANDEZ. We have identified him as a scientist in ORD.

Mr. MOLINARI. He works for ORD in EPA?

Dr. HERNANDEZ. Yes, headquarters area.

Mr. MOLINARI. In the documents that you furnished to the subcommittee earlier today, there is a memorandum dated May 1, 1981, sent to members of the Dow task force. It says, "Please review the final draft of the dioxin report by Milt Clark for technical accuracy and offer comments no later than May 15, 1981. As this report will be released to the public, it is extremely important that it be carefully reviewed."

Then, down at the bottom it says, "I have reviewed this document as to its accuracy. I have made some obvious necessary corrections, and against your wishes, unfortunately, had to editorialize in some places."

Now, I recognize the fact that this is a time when you were just coming onboard. You came on, as a matter of fact, after the date of this memorandum. But you served approximately 2 months when the final report was issued.

Can you or somebody else identify what role Mr. Rosius played in the changes that occurred in the final draft?

Dr. HERNANDEZ. I think there were a number of people in EPA who were called the Dow task force. It looks like there are a number of names on the letter.

Mr. MOLINARI. I assume they are all members of the Dow task force?

Dr. HERNANDEZ [conferring with staff]. The Dow task force is a region V group rather than a headquarters group. Region V sent this, evidently, to that group of people at headquarters to comment on too. The rest of these letters are from these people. So this is one by a scientist in ORD who said, "I reviewed it, and I made some editorial changes."

Mr. MOLINARI. I would hope there is somebody here who can tell us whether those editorial comments were made by Mr. Rosius in the margin.

Dr. HERNANDEZ. I would guess that is correct. I really do not know. The ink looks about the same.

Mr. MOLINARI. It does, indeed. You have one on PCDD's and PCDF's and fly ash and other combustion sources in the original draft report, and toward the bottom of the page in the margin, there is the word, "Bull," with an exclamation point.

Dr. HERNANDEZ. Yes.

Mr. MOLINARI. I know sometimes you test animals, but I suspect this word is the contraction of another word. There is a line alongside of the sentence which says, "The formation of PCDD's was attributed partly to the reaction between inorganic chloride and organic materials, and not due to the condensation of pre-existing polychlorinated phenyls."

Does anybody know whether that was Mr. Rosius's comment?

Dr. HERNANDEZ. I have no idea.

Mr. MOLINARI. Do you know what the final draft said and how it was changed with respect to that particular statement?

Dr. HERNANDEZ. No, sir. As I have said, I did not have anything to do with the final draft.

Mr. MOLINARI. Is there anybody here with you, Dr. Hernandez, who had anything to do with the final draft?

Dr. HERNANDEZ. I think Dr. Barnes had some input into that, but in terms of whether or not that was changed, I would guess there was probably a lot of give and take and the author may have looked at it and said, "Yes, I think you are right," or "I think you are wrong."

Mr. MOLINARI. Well, here is what I am getting at, and I hope that maybe somebody on your panel can offer some assistance. There have been numerous changes between the draft report and the final report, and I suspect that some of them may have been to correct inaccuracies. On the scientific exchange that took place, they wanted to put out a report that could stand scrutiny. I am interested in finding out precisely who the individuals were who were responsible for making the changes in the final report.

Dr. HERNANDEZ. I think we probably ought to do that. Mr. Molinari, something that would go along the lines of trying to identify what changes were made and the kind of rationale used.

Mr. MOLINARI. We have a conclusion in the draft report, which says, "The formation of PCDD's was attributed," et cetera and changed in the final report to read, "It was theorized that the formation," and then the language continues on. I suspect that whoever was responsible for making that amendment was questioning whether the conclusion was accurate or not.

Dr. HERNANDEZ. I think this is part of the problem. What is happening is they are taking a report written by a Dow Chemical person who made that conclusion, as I read it, and they say, "that was not a good conclusion," what you really need to do is go back—and that is why careful peer review, where somebody is totally responsible to make sure that references are read from the original text, to see whether or not it is what the author said.

Mr. MOLINARI. Well, Dr. Hernandez, some of the editorial statements in the margin indicate that material was cut, and that it was "Cut at Dow's request."

Dr. HERNANDEZ. I do not have any idea—and this is the draft that you have here, so Dow had not seen this at all.

Mr. MOLINARI. Well, somebody put in the margin, "Cut at Dow's request."

Dr. HERNANDEZ. Well, you know, this is the draft.

Mr. MOLINARI. Final draft or initial draft, the fact remains there is reference in the margin indicating that certain material was cut at the request of Dow Chemical.

Dr. HERNANDEZ. That is a very interesting thing. I had never noticed that. In fact, today is the first time I had seen this copy.

Mr. MOLINARI. Myself, as well.

Dr. HERNANDEZ. And that is an interesting comment. I am delighted you picked up on that. What this implies is that Dow may have seen this draft before I made it available to them.

Mr. MOLINARI. All right. I want to be fair with you. I do not want to question you on something you do not know.

Dr. HERNANDEZ. This draft was May 11 or May 8.

Mr. MOLINARI. Mr. Daniel, are you aware of any of this?

Mr. DANIEL. No, sir, I am not.

Mr. MOLINARI. Is there anybody at the table who was aware of it?

Dr. HERNANDEZ. Could we make sure for the record that we are on the same page and looking at the same sentence? Unfortunately, there are not page numbers on mine, but the top of the page starts "iii, Only trace amounts, if any, of 2-, 3-, 7-, 8-TCDD . . .," so for the record, whatever that page is——

Mr. MOLINARI. Yes.

Dr. HERNANDEZ. It is an interesting comment. I have no idea what it means. This is pre-Hernandez and Gorsuch as to the date, I believe, of this particular one, because if we would believe the date of Mr. Rosius's letter——

Mr. MOLINARI. Would you try to furnish for us a statement as to the changes that were made, who initiated those changes, how the final report appears, and who prepared that final report.

Dr. HERNANDEZ. We will try to do that for you.

Just one other clarification. Does your sentence have "Cut"?

Mr. MOLINARI. Does it have "cut"? Not there, no.

Dr. HERNANDEZ. OK. That's all.

Mr. MOLINARI. Take a look at "Summary of the Toxicological Effects of TCDD's and TCDF's on Humans," on page 8 which refers to a number of industrial accidents, some of which are rather famous, throughout the country and throughout the world, for that matter. Apparently, all of those studies were deleted.

Dr. HERNANDEZ. I really do not know. All I can say is——

Mr. MOLINARI. All right, then, why don't we continue with my original suggestion and have you make a review of the report for the subcommittee and furnish a statement which will indicate, number one, who made the recommendation as to specific changes and how those changes were implemented.

Dr. HERNANDEZ. To the extent that we can do this, we will. It is hard to try sometimes in those kinds of joint committees, because of the give and take sort of things that they do.

Mr. MOLINARI. I understand. Dr. Hernandez, one of the things that we have not touched on today but is a question that has been bandied about in the press lately is hit lists. There have been allegations that there are documents, or were documents, indicating that there are certain employees who were not held in favor because of their political leanings. Do you know anything about that?

Dr. HERNANDEZ. My first exposure that I have seen of the so-called hit list occurred when it became public from, I believe, Senator Hart, or Congressman Scheuer, I am not sure, and that was distributed around to people, and I did see a copy at that time.

Mr. MOLINARI. When was that?

Dr. HERNANDEZ. This was about 2 weeks ago or 1 week ago, something like that. I had not seen that document before. That document or any version like it played absolutely no role in the selection of any scientist or any EPA committee that I know of.

Mr. MOLINARI. Do you know whether there were employees of EPA who were being scrutinized because of their political leanings?

Dr. HERNANDEZ. I do not know that at all.

Mr. MOLINARI. Dr. Hernandez, did you ever go to the then known Mrs. Gorsuch and complain about the fact that some of your employees and people that were working with you had appeared on such a list?

Dr. HERNANDEZ. No.

Mr. MOLINARI. Mr. Daniels, are you aware of any such activities, or were you aware of any such activities?

Mr. DANIEL. I am not aware of any such activities.

Mr. MOLINARI. You knew of no hit lists?

Mr. DANIEL. I knew of no hit list. The hit list that was recently disclosed first came to my attention about March 1, after it had been released by committees here in Congress.

Mr. MOLINARI. Chip Wood allegedly is the individual who compiled at least some of the list. Who is Robert Herbolsheimer?

Dr. HERNANDEZ. He is a young man who works for me, Mr. Molinari, and takes care of both my travel arrangements and has had the kind of responsibility of moving paperwork through on these many EPA committees that we have, and was a repository of dozens of lists and recommendations that we got from many, many sources.

Mr. MOLINARI. According to the information I have, Dr. Hernandez, Mr. Herbolsheimer was questioned by Chip Wood about employee political affiliations. Do you know anything about that?

Dr. HERNANDEZ. I do not know about him questioned about employee relationships.

Mr. MOLINARI. Did there come a time when he came to you and made reference to conversations that he had with Chip Wood concerning the political background of himself or anybody else?

Dr. HERNANDEZ. Not himself, at all, ever. You know, Mr. Herbolsheimer came through the transition period, and when I came to EPA, I put him on my staff to take care of my travel and gave him this job of maintaining these files that we got from all kinds of people—the White House, the Congress, the Administrator, myself, SAB, other committee people.

Mr. MOLINARI. The information that I have received is that he was questioned, that he went to you, complained to you about it, and you thereupon took the information to Mrs. Gorsuch, and complained about the questioning of people who were working under you. Now, the question I have simply is that true or not true?

Dr. HERNANDEZ. Well, I think that the situation is a little bit different. It was not about people who were working under us or about people at EPA. It was an effort to scrutinize appointments that we were making to these committees which, while they may be EPA employees in some kind of a titular sort of way—that is, we pay them when they serve on the boards—they are not EPA employees in the sense of being 24-hour-a-day, 7-day-a-week persons like myself.

So in terms of EPA employees, the staff at that place, I do not have—Herbolsheimer had nothing to do with that kind of thing at all, nor did I, in terms of——

Mr. MOLINARI. As we look to the future, hopefully, we will see the restructuring of the Environmental Protection Agency, we will have gotten through these committee hearings, and you folks will have gotten back to the main task at hand. One of the impressions that I have received throughout the hearings is that there was a great deal of looking over your shoulder. People were working, and there were other employees who were making reports on those people. Indeed, we have the infamous memo that made references to Mr. Perry. It seems as though the atmosphere at the Environmental Protection Agency was one where the professionals might have had some difficulty working because of the cloud that was raised about who was really calling the shots.

Is it fair to say that you had a serious morale problem?

Dr. HERNANDEZ. Well, I think that that probably would be an understatement, and in some aspects, it has deteriorated. But I believe in all sincerity that things have gotten a little bit better, and given that we have some breathing room—you know, when we come up every day and are questioned on philosophy about whether or not you went ahead on this or that, you know, those are not philosophical issues; those are judgment calls on things, and gosh, you use the best you can. And to make each one of these the subject of a congressional investigation just does not merit it. I hope we would get down to letting us work and deal with these problems.

Mr. MOLINARI. I can understand your concern, and I share that concern. Look at the array of witnesses that we have and the people behind you. These are people who should be back in the shop, doing some work. This is one of my serious concerns.

Dr. HERNANDEZ. Particularly this guy.

Mr. MOLINARI. Particularly Mr. Hedeman, yes. But by the same token, the hearings have proved to me that there are some changes required at the Environmental Protection Agency, both in terms of personnel and in terms of policy. Certainly, I think this subcommittee will take a back seat to nobody in our overview of that process and the Agency. We look forward to working with you folks. I have got a great deal of respect for a number of you out there who have helped a great deal.

We have to achieve a better spirit of cooperation between EPA and Members of Congress and we must share information. We would not be here today, I do not think, nor would, perhaps, the other committees and subcommittees be involved, if we did not have those original stone-walling attempts. We had an inquiry that was going very nicely, initiated by myself—I must say, as far as this subcommittee was concerned. We had a great spirit of cooperation, and then suddenly, the wall appeared, and it led to all these Congressional inquiries.

I really do not think the fault lies on this side of the table, but rather, with EPA, perhaps the White House, and certainly, the Justice Department has to bear their share of the blame.

Dr. HERNANDEZ. Mr. Chairman, Mr. Molinari, I agree with you. The agreement that we struck the day I came sounded like a fair and open process. Unfortunately, I did not know about it being terminated, and the introduction of the Justice Department's concern that we had to treat all administrative documents, enforcement documents, administration documents, like this, and it was no longer an opportunity for John Hernandez to deal with Congressman Molinari and the chairman. So I really hope to reestablish that kind of rapport that we did share, and I need to do it with all of the committees.

Mr. MOLINARI. I understand that. I thank you very much, Dr. Hernandez, and the other people who are here today from EPA.

Mr. Chairman, I have no further questions.

Dr. ROWLAND [presiding]. The gentleman from Michigan?

Mr. ALBOSTA. Thank you, Mr. Chairman.

Dr. Barnes, I would like to ask you a couple of questions. You indicated earlier, Dr. Barnes, that you taught at a college in North Carolina for 12 years, is that correct?

Dr. BARNES. Yes; that is correct.

Mr. ALBOSTA. Prior to assuming your position with EPA—and I would assume you had no other position between that time——

Dr. BARNES. That is correct.

Mr. ALBOSTA [continuing]. While teaching at that college or prior to that, did you ever perform consulting assignments?

Dr. BARNES. I came to EPA on a sabbatical in, I think, 1977-78 and following that sabbatical, in the ensuing academic year, I served as a consultant to the Agency.

Mr. ALBOSTA. Did you ever serve as a consultant to anyone in the chemical industry or any other manufacturing company?

Dr. BARNES. No; not that I am aware of. I do not recall any at all.

Mr. ALBOSTA. Those were my primary questions to you.

Dr. Hernandez, what are your thoughts now? What are we going to do in Michigan. The State of Michigan has applied for $450,000 in grants so it can monitor dioxin pollution in the fish in the Michigan rivers. Are you going to go ahead as fast as possible to see that that grant is given to the State?

Dr. HERNANDEZ. I do not know where the grant is. I guess it is in the regional office. If it is a reasonable proposal that has a reasonable opportunity for a good outcome in a timely fashion, and that we need that kind of information—somebody may claim that we've got a good base right now—then, we should fund the proposal.

Let me tell you what I think is a good outcome of this.

Mr. ALBOSTA. I would like to hear it.

Dr. HERNANDEZ. I think that one of the things that we might do—we are into a litigation with Dow and the Justice Department at this point—if there has been runoff from the Dow plant—and these TCDD's, dioxins, do adhere to soil particles; they are tenacious on those things—one of the places we ought to look is out in the river, and I guess the Tittabawassee is right off of Dow——

Mr. **Albosta**. It is right between its plant.

Dr. **Hernandez** [continuing]. Yes, looking in some of those sediments at the relative concentrations there and some kind of plume away from there, I think that would be a valuable kind of study. Now, I do not know that we have done that or not, but that would be of use. And if the State of Michigan came forward with something like that, a good proposal on how to do that sort of thing, that would be one of the approaches, because I think you need to establish a source and a mode of transport and an end result. It was implied by looking at the levels of dioxin in fish that, in fact, Dow was the cause. We need to pin that down. We need to make that decision, find that information.

Mr. **Albosta**. No one could agree with you more than I; I can tell you that, because I do hope that we find whatever the source is and whoever is responsible, and that we are able to somehow identify that and to bring some kind of clean-up procedures.

Dr. **Hernandez**. That would tie well with what the region is doing, because theirs is on an NPDES permit—that is discharging a liquid waste out there—and this would tie well with what they have done.

Mr. **Albosta**. I do not like the fact that Michigan is constantly having problems, either environmentally or whatever, and that we have problems trying to find jobs for our people. And I do not want any industry that tries to do a good job to move out. But I also do not want my people dying because we did not do something or we did not regulate some procedure. It all goes hand in hand, and we have to do it, and we have to do it together, and I am sure that we cannot exist if we do not have some of the products that are made. We know it is dangerous today to produce a lot of these toxic chemicals. We want to know the best possible methods to do it—I do, at least, and I want to know how we are going to dispose of the waste that comes from the production of those chemicals and what is the best procedure to do it.

Would you be in favor of some type of changes in the tax laws to give credit to those companies like Dow Chemical Co., that have the production of the feedstocks and now, most of the cost of the responsibility of the Superfund moneys? Would you think that some arrangement ought to be worked out so that in the future, we can say to them—let me finish on this, so you know precisely what I am trying to get at—if, let us say, the Dow Chemical Co. could find with EPA's approval, and with other environmental groups' approval, a way to dispose of this material through furnaces, high temperature or whatever, and that it met all the standards and so forth, would a tax credit from the Superfund be the best interest at that particular time? Wouldn't that encourage some future, high-technical way, perhaps, being found of disposing of this without making more problems for the next generation, or two or three or four generations down the line, where we have no idea, in some cases, of what might be and might somehow leak out and cause damage and how it might get into the environment again.

I was very much involved in Michigan during the incident of PBB. It was spread almost evenly through the whole State. No enemy of ours could have ever, I do not believe, found a way to so evenly distribute a toxic chemical to every one of the residents of our State—almost, except for the western part of the upper peninsula.

It just seems to me that we are asking for trouble, even though we may be trying to seal these sites in and so forth; down the road somewhere, we are asking for trouble, if we do not try to put together the best technology now possible. And I have had in mind a way of trying to relieve some of the costs on those companies if they would provide some of the necessary technology and the unit itself to dispose of that material in the future, or maybe some of it that is in these sites now, that could be brought in and disposed of in an orderly fashion.

Your comments on that.

Dr. HERNANDEZ. I think any time we can provide people with an incentive to do these things, we are better off, because there is a driving force out there that says, "Solve a problem," and somehow that cost is absorbed and not passed on; it is part of that taxing system.

I would not like to see the Superfund tax affected by some kind of a tax credit. I would like to see what we are doing now maintained until decisions are made in the 1984 period about continuation, or whatever is the next step down the line on this.

Mr. ALBOSTA. It would seem to me, with already identifying 14,000, I believe the number is, waste sites, that we have not seen the end of it. And if we do not do something now to correct the problems that we are trying to catch up on with 14,000, maybe 20,000 of them, we will have maybe 20,000 of these sites somewhere in the future. And that is the question we have to look at. No matter what the cost is in this particular case, we ought to have an incentive to those companies to put their technology to use so that we do stop it once and for all, and we assure the American people that we are doing something, and we are concerned about their health needs and their requirements in the environment and what is in the best interest of all of us.

Dr. HERNANDEZ. Sounds like a——

Mr. ALBOSTA. Mr. Chairman, I have one more request. I would like to enter into the record a letter I sent to Doug Costle back on March 7, 1979 that has gotten out. I was asking Mr. Costle at that time to give me an explanation of the points that I raised in the letter. I would like to enter this letter into the record and make it part of the record, because I do not like the idea that somebody is getting this letter without knowing full well what the intent of it was.

Mr. LEVITAS [presiding]. Without objection, so ordered.

[The following was received for the record:]

SPECIAL

Congreſs of the United States
House of Representatives
Washington, D.C. 20515

March 7, 1979

SPECIAL

OTS/3/17
cc: DMC
cc RWS
Jellinek

Mr. Douglas Costle, Administrator
Environmental Protection Agency
401 M St.,S.W.
Washington, D.C. 20460

Dear Mr. Costle:

It has recently come to my attention that you have issued emergency suspension orders for the herbicide 2,4,5-T.

There are three fundamental points that I would like to make about this matter.

1) As the Congressman representing the district in which a large percentage of the product is manufactured, I believe that I deserve more than a few hours notice before such drastic action is taken.

2) The new information used as a basis for emergency removal of 2,4,5-T from the market does not appear to be sufficient to warrant abandoning the regular procedure for hearings under the Rebuttable Presumption Against Registration provisions. Therefore, I believe this action to be arbitrary and capricious.

3) I hope this action does not represent a change in standards required for emergency agency action.

I would appreciate an explanation of your policy on the points raised here.

Sincerely,

Don Albosta

Don Albosta
Member of Congress

DA:jf

Mr. LEVITAS. Dr. Hernandez, there are some very important additional areas that we want to explore, but as I had committed, we are going to try to take a break. I am sorry it has gone as long as it has without any interruption. Then we can get back.

Dr. HERNANDEZ. Shouldn't we just work on through—maybe just a standup break of 10 minutes or so and come back?

Mr. LEVITAS. That will be fine I just think you ought to have an opportunity to just sort of stretch your legs for a little while. I was informed that there was a desire for a break.

Dr. HERNANDEZ. People probably need a short one. You know, if we say 10, we will be back in 15. Let's try 10?

Mr. LEVITAS. Well, it is 10 minutes of 2. The subcommittee will stand in recess for 10 minutes, and when we reconvene, Mr. Molinari has some follow-up questions, and then Dr. Rowland and myself.

While we are at the break, I want you to think about that Dallas business we were talking about, because I consider that to be one of the most serious issues that has arisen in this whole matter up to this point. Maybe you can get some more information about it.

The subcommittee will stand in recess until 2 p.m.

[Short recess.]

Mr. LEVITAS. The subcommittee will please come to order.

The Chair recognizes the gentleman from New York.

Mr. MOLINARI. Thank you, Mr. Chairman.

During the recess, in a conversation I had with some of the staff from EPA, the possibility exists that the editorial comments that appeared in the margins of the documents that we have with respect to Dow Chemical and the dioxin report may have indeed been added by members of one of the other subcommittees, conducting their own inquiries. In a spirit of fairness, I want to just make that statement in the record. The copies that I have received have those comments and I assume that those were comments that were on the documents before any congressional committee received them. We of course will continue to investigate but I did want to mention that it has been brought to my attention that the copies that Dr. Hernandez has and the rest of the staff from EPA, do not have those notations in the margin. I want to make that statement for the record and then we will pursue it at a later time.

Do you want to make some comment on that, doctor?

Dr. HERNANDEZ. I think you're correct. I believe the copy you have is the one I brought with me today, was Xeroxed here and circulated. I think you're absolutely correct on it; that those comments were made by somebody at EPA headquarters or somebody in the region; it's hard to tell which.

Mr. Chairman, I wonder if we might submit for the record a copy of the final report, that July 1981 report, plus the paper that is appended to it that is part of it, called "The Long Term Hazards of Polychlorinated Dybenzodioxines and Polychlorinated Dibenzofurans."

Mr. LEVITAS. Without objection, the final report and the appended item will be made part of the record, and also a copy of the Dallas Area Lead Study. I think it is February 1981. And it will likewise be made a part of the record, without objection.

[The following was received for the record:]

A REPORT ON POLYCHLORINATED DIBENZO-P-DIOXINS (PCDDs) AND POLYCHLORINATED DIBENZOFURANS (PCDFs): A SUMMARY OF STUDIES CONDUCTED IN THE GREAT LAKES AREA

U.S. ENVIRONMENTAL PROTECTION AGENCY
REGION V
CHICAGO, ILLINOIS

July 1981

TABLE OF CONTENTS

List of Tables.. ii

I. Introduction.. 1

II. Sources of Polychlorinated dibenzo-p-dioxins (PCDDs) and dibenzofurans (PCDFs) in Commercial Products................ 2

III. Sources of PCDDs and PCDFs from Pyrolysis, Photolysis, and Combustion Processes.. 2

IV. Studies Involving PCDDs and PCDFs Conducted in the Great Lakes Area.. 10

V. Current Government Activity on PCDDs and PCDFs................. 24

References.. 25

LIST OF TABLES

Table 1 - Findings on PCDDs and PCDFs in Fly Ash and Other Combustion Sources.. 6

Table 2 - Chlorinated Dioxin Content of Particulate Matter From a Rotary Kiln Incinerator............................ 7

Table 3 - Chlorinated Dioxins in Rotary Kiln Incinerator Scrubber Water.. 8

Table 4 - Chlorinated Dioxins in Soils and Dusts.................... 9

Table 5 - Results of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) Fish Monitoring Program Conducted by the U.S. EPA in Michigan during 1978.......................... 14, 15, 16

Table 6 - Residues of Dibenzofurans and Dibenzo-p-dioxins in Composite Samples of Fish and Gulls from Different Sites on the Great Lakes.................................... 21, 22

Table 7 - Samples Analyzed from Crete Metals, Crete, IL............. 24

LIST OF FIGURES

Figure 1 - Saginaw River Basin....................................... 17

Figure 2 - Dioxin in Gull Eggs....................................... 19

I. Introduction

In the last several years, concern over the environmental and public health effects from the use of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and Silvex, plant herbicides, has increased. Chemical products containing 2,4,5-T have been shown to contain a highly toxic trace contaminant, 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD), which is considered to be the most toxic of the 22 possible isomeric forms of TCDD. On February 28, 1979, an emergency suspension was imposed on certain uses of 2,4,5-T and Silvex based on extensive animal data showing that exposure to 2,4,5-T, Silvex and/or 2,3,7,8-TCDD resulted in fetotoxic, teratogenic, and carcinogenic effects and on selected epidemiological data (1,2). TCDDs and other chlorinated dioxins, technically known as polychlorinated dibenzo-p-dioxins (PCDDs) and related compounds, polychlorinated dibenzofurans (PCDFs) are found in certain commercial products. Those compounds which have a broad range of toxicities, are also receiving considerable attention. Evidence exists that PCDDs and PCDFs may be released to the environment by combustion processes. This report summarizes those studies involving PCDDs and PCDFs in the Great Lakes Area. This report does not address the physical, chemical, and toxicological properties of these compounds. Several extensive reviews on the properties of PCDDs and PCDFs are available (3,4,5,6). One of these reviews, "Long Term Hazards of Polychlorinated Dibenzodioxins and Polychlorinated Dibenzofurans," by Huff, et al is attached at the end of the report.

II. Sources of Polychlorinated dibenzo-p-dioxins (PCDDs) and dibenzofurans (PCDFs) in Commercial Products

A. 2,4,5-T and Silvex (phenoxy herbicides)

2,3,7,8-TCDD is formed in the manufacture of 2,4,5-trichlorophenol from 1,2,4,5-tetrachlorobenzene, especially if production temperatures exceed 160ºC(5). Drums of Agent Orange, a mixture of 2,4,5-T and 2,4,-D, stored in the USA and in the Pacific prior to 1970 have been reported to have 2,3,7,8-TCDD levels ranging from 0.1 to 47 ppm (7). Only trace levels of other PCDDs and PCDFs were found. In 1978, producers of 2,4,5-T reported that their products contain less than 0.1 ppm of 2,3,7,8-TCDD (5).

B. Chlorinated Phenols

PCDDs and PCDFs have been found in tri, tetra, and pentachlorophenols (4,8,9). The quantities of these compounds and their isomeric forms depend upon the reaction process and conditions. In 1978 concentrations of 2,3,7,8-TCDD in 2,4,5-trichlorophenol and its sodium salt were found to be less than 0.1 ppm (10). Analyses of PCDDs in commercial grade pentachlorophenols (PCP) produced by various manufacturers during the years 1975 to 1977 found hexa-CDDs (HxCDDs) (non-detectable (N.D.) to 520 ppm), hepta-CDDs (HpCDDs) (125 to 180 ppm) and octa-CDDs (OCDDs) (N.D. to 3600 ppm). No TCDDs were found in commercial PCP. There were several isomeric forms of HxCDD, the major isomers being the 1,2,3,6,8,9; 1,2,3,6,7,8; and 1,2,4,6,7,9. The isomeric forms for HpCDD were 1,2,3,4,6,7,9 and 1,2,3,4,6,7,8(8). PCDFs were also detected in PCP including hexa-CDFs (HxCDFs) (9 to 90 ppm), HpCDFs (70 to 400 ppm) and OCDFs (55 to 260 ppm).

C. Other Chemicals

PCBs have been shown to contain 1-10 ppm of various PCDFs (11). Hexachlorophene has been reported to contain traces of TCDDs (4).

III. Sources of PCDDs and PCDFs from Pyrolysis, Photolysis and Combustion Processes

A. Chlorophenols and Related Compounds

The pyrolysis or combustion of chlorophenols, chlorophenoxy compounds or their derivatives has been shown to produce various PCDDs including TCDDs. The 2,3,7,8 isomer of TCDD has been detected in some experiments although generally as a minor component of the total TCDDs and PCDDs. Studies conducted on the burning of materials such as grass, leaves or wood chips that have been treated with chlorophenols or related compounds have also demonstrated that PCDDs are formed (7,12-16).

B. Formation of PCDFs by Pyrolysis

1. The pyrolysis of commercial PCBs (Aroclor 1254 and 1260) at temperatures of 500-600°C has been shown by Buser and his co-workers to yield 30 major and more than 30 minor PCDFs. From 3 to 25% of the pyrolyzed PCB was converted to PCDFs. Above 700°C only trace amounts of PCDFs are formed due to the complete destruction of PCB. The formation of PCDFs was shown to be the result of intermolecular cyclizations of PCB (11).
2. PCDDs and PCDFs are produced from the pyrolysis of tri, tetra, and pentachlorobenzenes in the presence of air. TCDDs have been found in pyrolysates of trichlorobenzene. However, the 2,3,7,8-TCDD isomer was present only as a minor constituent (12).
3. Recently it was reported that soot from the burning of transformer fluids containing 65% PCB and 35% chlorinated benzenes, mainly trichlorobenzene and tetrachlorobenzene, contained 3 ppm of 2,3,7,8-TCDD and 273 ppm of 2,3,7,8-TCDF (15).

C. Formation of PCDDs by Photolysis

Dechlorination of OCDD in pentachlorophenol produces tetra, penta, hexa and hepta chlorinated dioxins (8,17).

D. PCDDs and PCDFs from Combustion Sources and in Soil and Dust Samples

1. Total amounts of PCDDs and PCDFs in fly ash from a municipal incinerator in Switzerland were found to be 0.2 ppm and 0.1 ppm respectively. At an industrial heating facility, the fly ash contained 0.6 ppm PCDD and 0.2 ppm of PCDF. Only traces of the more toxic PCDDs (2,3,7,8; 1,2,3,7,8; 1,2,3,6,7,8; 1,2,3,7,8,9) were detected (7). More recent comprehensive studies have shown that PCDDs and PCDFs were present in the fly ash of 35 municipal incinerators in Europe up to 1 ppm amounts (4,14). The formation of PCDDs and PCDFs are thought to be from the combustion of wastes containing polychlorinated phenates related compounds (15,16).

2. A study released by Dow Chemical in 1978 and published in 1980 (18) reported no 2,3,7,8-TCDD (10 ppb detection level) in fly ash from their oil and coal power plant; however, 38 ppb of other TCDD isomers were found at a detection limit of 20 ppb. Detectable levels of PCDDs were found in samples from fireplaces, automobile and truck mufflers and cigarette smoke. The authors concluded that dioxins have been present since the advent of fire. The results of their study are presented in Table 1 on page 6. In contrast to the 1978 report, the authors in their 1980 article tempered their remarks by stating that some combustion sources may be sources of airborne and waterborne particulates containing PCDDs. It was theorized that the formation of PCDDs could be the result of a reaction between inorganic chloride and organic material and not due to the condensation of preexisting polychlorinated phenols. The 1980 report also included

data on emissions from a rotary kiln incinerator operated by Dow Chemical and analyses of soils and dusts collected between 1976 and 1978. Particulate matter collected from the rotary kiln incinerator, operated without natural gas and burning solid wastes from the facility, had levels of total TCDD reaching 13,200 ppb (Table 2). Two of the samples collected had levels of the 2,3,7,8-isomer greater than all other TCDD isomers. No TCDD emissions were noted when the facility was operated with natural gas. Other PCDDs were found to be substantially reduced (>95%). Analyses were also performed on particulates filtered from the scrubber water (Table 3). Levels of 2,3,7,8-TCDD and other isomers were reduced by 90% when the kiln was operated with natural gas. Results of soil and dust samples reported by the authors are presented in Table 4. The highest levels of PCDDs in soil and dust samples were those collected at the Midland plant and could be from combustion sources and/or chlorophenolic production by Dow Chemical. Evaluation of all data reported has been made by EPA personnel (19,20).

3. The results of Kimble and Gross (21) contrasted with the Dow Chemical findings. At one coal fired power plant they found no TCDD at a detection limit of 1.2 ppt in fly ash. Based upon work in their laboratory Lustenhouwer, et al, (14) have questioned the extraction method used by Kimble and Gross; however, the method was not exactly duplicated (43). In addition, the apparently negative results of Kimble and Gross have subsequently been corroborated by EPA from tests on fly ash obtained from seven coal-fired power plants (44,45).

TABLE 1

PCDDs and PCDFs
In Fly Ash and Other Combustion Sources (Reference #18)

Sample	Number of samples	Apparent dioxin content, ng/g(ppb)					
		TCDD					
		Other isomers	2,3,7,8-	Total	HCDD	HXCDD	OCDD
Incinerators, powerhouse							
• Dow powerhouse	1	38(20)	N.D.(10)	38(20)	2	4	24
• Dow rotary incinerator stack (present normal operation with supplemental fuel)	5			N.D.(2)	1-5	4-100	9,950
• Dow stationary tar burner stack (normal operation with supplemental fuel)	5	N.D.	N.D.	N.D.	1-20	27-160	190-440
• U.S. municipal incinerator (electrostatic precipi tator)(Nashville, TN)	1	7.3	0.4	7.7	14	28	30
European municipal incinerators				2-20	30-200	60-130	40-120
Mufflers							
Diesel truck muffler	2	0.02	0.003	0.023	0.020	0.100	0.26
Auto muffler	4	N.D.-0.004	N.D.-0.004	N.D.-0.008	N.D.	0.003-0.01	0.02-0.07
Other sources							
Home fireplace soot	2	N.D.-0.3	N.D.-0.1	N.D.-0.4	0.2-3	0.7-16	0.9-25
Home electrostatic precipitator	1	0.4(0.4)	0.6	1.0	34	430	1,300
Cigarette smoke	2	N.D.	N.D.	N.D.	0.004-0.008	0.009	0.02-0.05
Charcoal-broiled steak	4	N.D.	N.D.	N.D.	N.D.	N.D.	0.03(0.03)

Where multiple samples were analyzed from similar sources, the range of observed values is shown. 2) N.D. indicates that the signal observed was less than 2.5 times noise. Limits of detection not within these ranges are shown parenthetically after the value reported for signals between 2.5 and 10 times noise. 3) Data without supplemental fuel is not included here because this practice has been eliminated.

TABLE 2

Chlorinated Dioxin Content of Particulate Matter From a Rotary Kiln Incinerator

(Reference #18)

	Fuel[1]		Apparent dioxins, ng/g (ppb)				
Sample	Kiln	Secondary	Other isomers TCDD	2,3,7,8-TCDD	HxCDD	H_pCDD	OCDD
			Without supplemental fuel				
R_1F_1	T,SW,G	T	1,800	2,800[2]	13,000	110,000	180,000
R_1F_2	T,SW,G		5,000	8,200[2]	65,000	510,000	810,000
R_2F_1	T,SW,G		3,300	110	1,300	2,000	3,000
R_3F_1	T,SW,G		12,000	N.D.(260)[3]	5,600	37,000	59,000
			With supplemental fuel				
R_3	T,SW,G	O,G	N.D.(8.0)	N.D.(2.0)	1.4	13.0	30.0
R_4	T,SW,G		N.D.(7.0)	N.D.(5.0)	N.D.(1.0)	4.0	9.0
R_5	T,SW,G		N.D.(2.0)	N.D.(2.0)	N.D.(0.5)	6.0	15.0
R_6	T,SW,G	T,G	N.D.(2.0)	N.D.(4.0)	5.0	27.0	170.0
R_7	T,SW,G		N.D.(2.0)	N.D.(2.0)	4.0	110.0	950.0

1. T, tars; SW, solid waste; G, gas; O, oil.

2. The high results reported for the 2,3,7,8-isomer are probably due to analysis by the nonspecific GC-MS packed-column method. Later results were obtained with a capillary column specific for 2,3,7,8-TCDD. The TCDD results from run 1 (R_1) are not comparable to those from runs 2 and 3.

3. Numbers in parentheses are limits of detection.

TABLE 3

Chlorinated Dioxins in Rotary Kiln Incinerator

Scrubber Water

(Reference #18)

Sample	Apparent dioxins, ng/g (ppb)				
	Other isomers TCDD	2,3,7,8-TCDD	HxCDD	H_pCDD	OCDD
Particulates filtered from scrubber water					
Without supplemental fuel	300	2,200 *	3,400	26,000	42,000
With supplemental fuel	14	32*	200	970	1,200
Filtered scrubber water without supplemental fuel	0.0018(0.01)**	0.001*(0.0006)**	0.005	0.024	0.026

* The analytical method did not separate 2,3,7,8-TCDD from 11 other isomers.

** Numbers in parenthesis are limits of detection.

TABLE 4

Chlorinated Dioxins in Soils and Dusts (Reference #18):

	Number of samples	Apparent dioxin content, ng/g (ppb)[1]					
		Other isomers TCDD	2,3,7,8-TCDD	Total TCDD	HxCDD	HpCDD	OCDD
Soil							
Rural (Gaylord, MI)	5			N.D.[2]	N.D.	N.D.-0.05	N.D.-0.2
Urban (Lansing & East Lansing, MI)	5			N.D.	0.03-1.2	0.03-2	0.05-2
Major metropolitan (Chicago, IL)	8			0.005-0.03	0.03-0.3	0.1-3	0.4-22
Dow Chemical (Midland, MI)	5	0.8-18	0.3-100	1-120	7-280	70-3,200	490-20,000
Dust							
Dow Chemical lab.	6	0.5-2	0.7-3	1-4	9-35	140-1,200	650-7,500
Midland, Michigan	2			0.03-0.04(0.02)[3]	0.2-0.4	2-4	20-30
Metropolitan (Detroit, MI)	4			N.D.-(0.03)	N.D.-0.3	0.3-4	0.1-4
Metropolitan (St. Louis, MO)	1	0.16	0.12	0.3	2	34	210
Metropolitan (Chicago, IL)	2			0.04(0.04)	N.D.-0.3	0.6-3	3-8

1. Where multiple samples were analyzed from similar sources, the range of observed values is shown.
2. N.D. = Not Detectable
3. Value in parentheses indicates detection limit.

IV. Studies Involving PCDDs and PCDFs Conducted in the Great Lakes Area

A. Dow Chemical Monitoring Study, Midland, Michigan (June, 1978)

In June, 1978, Dow Chemical reported results of their monitoring program to the Michigan Department of Natural Resources (MDNR) as requested by that agency (22). The data has been summarized below.

1. Tittabawassee River Water and Sediment (1976)

Ten samples (two water and eight sediment) from the Tittabawassee River and below the plant showed no detectable amounts of TCDDs. The detection limit (DL) for sediments ranged from 10 to 50 ppt. Water samples were analyzed at a DL of 1 ppt.

2. Plant Discharge Stream (1976-1978)

Composite and grab samples of wastewater discharge revealed no detectable levels of TCDDs in 13 of 14 samples (DL=5 ppt or less). One sample was found to contain 8 ppt of TCDDs.

3. Bioconcentration Study (1978)

Analyses performed on the edible portions of fish flesh from caged trout placed six miles downstream of the plant's effluent for periods of 7,14, and 30 days revealed no detectable level of TCDDs at a DL of 10 to 30 ppt. Five of six fish placed in the tertiary effluent stream for 7 days were found to have TCDDs ranging from 20 to 50 ppt at a DL of 10 to 20 ppt. When analyses were also performed on whole fish, the 30 day trout (downstream) showed TCDDs ranging from 10 to 20 ppt when analyzed at a DL of 5 ppt. The fish collected from the tertiary effluent stream showed higher levels of TCDDs ranging from 50 to 70 ppt at a DL of 20 ppt. Analyses on the 7 day trout (edible and whole fish) caged along the river bottom revealed no TCDDs at a DL of 10 to 40 ppt. No TCDDs were detected in analyses performed on the edible portions of fish flesh from unexposed control fish (DL=20 ppt) and those placed in the river at Dow dam (DL=20 ppt), indicating that TCDDs could be present in the plant's effluent (4).

4. Native Fish Studies

(a) 1976 (Analyses 1978)

TCDDs were detected in 4 of 9 catfish samples, edible portions, at levels ranging from 70 to 230 ppt. Three of the four samples in which TCDDs were detected were located downstream of the plant's effluent. OCDDs were found in 8 of 9 fish, collected both upstream and downstream of the discharge. The levels ranged from 40 to 150 ppt. One fish sample was positive for HxCDDs (90 ppt). Detection limits ranged from 10 to 200 ppt.

(b) 1977 (Analyses 1978)

Fish caught downstream of Dow at Smith's Crossing (Figure 1) in the Tittabawassee River revealed contamination in 9 of 14 fish tested. Analyses were performed on the edible portions of fish flesh. The levels of TCDDs ranged from 20 to 244 ppt.

(c) Saginaw Bay 1978

Of three fish collected from Saginaw Bay, one catfish was found to contain 24 ppt TCDDs, at a DL of 10 ppt. The analyses were performed on the edible portions of fish flesh.

5. Chlorophenols

Chlorophenols were found in the Dow effluent up to levels of 70 ppb. 2,3,4,6-tetrachlorophenol and pentachlorophenol were present at ppb levels below the dam in sediments of the Tittabawassee River. Chlorophenols were also found in some fish at ppb levels above the discharge point of the plant's wastewater effluent.

Based upon these findings, the Michigan Department of Public Health issued an advisory on June 28, 1978 against consuming fish caught in the waters of the Tittabawassee and Saginaw Rivers. This health advisory is still in effect at this date (23).

B. Dow Chemical Monitoring Study, Midland, Michigan (October, 1978)

Results of PCDD sampling indicated the presence of 2,3,7,8-TCDD in 2,4,5-T sludge at 10 ppt and in trichlorophenol scrubber fluid at 20 ppt (24). The 2,4,5-T sludge also contained 11 ppt HxCDDs, 4 ppt HpCDDs, and 43 ppt OCDDs Brine wastes and sludges from 2,4-D processes were negative for PCDDs.

C. U.S. EPA TCDD Fish Monitoring Program (December, 1978)

In response to Dow's first monitoring report, which indicated incidental contamination of fish from the lower Tittabawassee River, fish were collected and analyzed from the Tittabawassee, Grand and Saginaw Rivers (25). In 26 out of 35 skinless fillet fish samples, detectable levels of 2,3,7,8-TCDD were found and ranged from 4 to 695 ppt. Eleven of these fish samples contained TCDD levels greater than 40 ppt. All but two of these samples were from locations downstream of Midland. Four out of 6 samples collected upstream were found to be negative for 2,3,7,8-TCDD. Data is presented in Table 2 on pages 14 and 15. Analyses of spiked blind controls, which consisted of various known levels of 2,3,7,8-TCDD, indicated that the positive results were within ± 20% of the values reported. Responding to a request from the Governor of Michigan, the FDA recommended that fish containing TCDD (2,3,7,8-TCDD) at a level of more than 100 ppt not be consumed. Where 2,3,7,8-TCDD had been found in fish below 100 ppt, it was recommended that no more than one average size portion of fish be consumed per week (26).

D. U.S. EPA-Michigan Monitoring Study of Dow Chemical, Midland, Michigan (April, 1979)

Results of TCDD analysis of samples taken from Dow's 2,4,5-Trichlorophenol injection well (DL=0.06 ppt), phenol treatment system (DL=82 ppt), plant effluent (DL=0.002 ppt) and upstream Tittabawassee River (DL=5.0×10^{-5} ppt), were found to be negative. However, the carbon filter concentration system utilized was believed to be unsuitable for concentrating contaminants found in water (27).

E. Dow Chemical Study on Fish, April 1979

As part of Dow's study on dioxin the company reported finding the following ranges of dioxins in parts per trillion (ppt) in fish collected from other tributaries of the Saginaw River (28).

	Number of Samples	Number of Positive Results	TCDDs	HxCDDs	HpCDDs	OCDDs
Flint Fiver	6	5	ND-11	ND-6	ND-5	ND-20
Cass River	6	4	ND-104	ND-11	ND-11	ND-44
Shiawasse River	6	1	ND-9	ND	ND	ND

ND=not detected

F. Composite Fish Sample from the Tittabawassee River (December 1980)

A single composite whole fish sample of five carp, supplied by the MDNR, (collected downstream of Midland) and analyzed by Columbia National Pesticide Laboratories, yielded positive results for various PCDDs and PCDFs (29). The 2,3,7,8-isomer of TCDD constituted >90% of the TCDDs found (30,31). The results are shown below:

	TCDDs	81	TCDFs	37
	PCDDs	31	PCDFs	73
	HxCDDs	44	HxCDFs	145
	HpCDDs	53	HpCDFs	31
	OCDDs	14	CCDFs	4
Total	PCDDs	223 ppt	Total PCDFs	290 ppt

TABLE 5

RESULTS OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN (TCDD) FISH MONITORING PROGRAM CONDUCTED BY THE U.S. ENVIRONMENTAL PROTECTION AGENCY IN MICHIGAN DURING 1978

LOCATION	FISH SPECIES	FISH LENGTH (cm)	* TCDD LEVEL (ppt)[a]	TCDD DETECTION LIMITS (ppt)
Tittabawassee River				
Tittabawassee Road	Carp	42.2	52	8
	Yellow Perch	16.2 11.0[b]	20	8
	Carp	39.7	93	6
Freeland Road	Carp	40.0	32	4
	Yellow Perch	16.8 12.3	10	5
Smith's Crossing Road	Channel Catfish	50.3	273	6
	Carp	38.0	22	11
	Channel Catfish	38.2	695	60
	Carp	23.1	49	1
	Sucker	35.6	8	4
	Sucker	38.1	21	4
Above Dow Dam	Channel Catfish	30.9	42	9
	Carp	34.6	ND[c]	5
	Carp	33.0	ND	9
	Channel Catfish	38.1	28	2
	Yellow Perch	19.3	ND	4
Dublin Road	Carp	52.2	ND	9

* Skinless Fillets

TABLE 5 (cont'd)

LOCATION	FISH SPECIES	FISH LENGTH (cm)	* TCDD LEVEL (ppt)a	TCDD DETECTION LIMITS (ppt)
Saginaw River				
Wickes Park	Carp	46.0	62	13
	Yellow Perch	11.9 11.4	ND	11
Block's Marina	Channel Catfish	69.0	105	7
	Channel Catfish	48.5	52	8
	Carp	51.8	28	4
Mouth	Channel Catfish	46.0	30	6
	Carp	45.0	153	13
	Yellow Perch	16.0 16.0	11	4
Grand River				
Jones Road near Eagle	Carp	39.0	20	4
	Carp	39.0	41	8
	Channel Catfish	----	29	6
	Smallmouth Bass	35	7	6
	Smallmouth Bass	38.0	8	6
Near Ionia	Carp	50.8	ND	2
	Sucker	48.3	ND	2
Saginaw Bay				
Lat. 43 40' 16"	Sucker	28.2 31.0	4	2
Long. 83 50' 21"	Yellow Perch	24.2 26.8	ND	3

* Skinless Fillets

TABLE 5 (cont'd)

Location	FISH SPECIES	FISH LENGTH (cm)	* TCDD LEVEL (ppt)[a]	TCDD DECTECTION LIMITS (ppt)
Lake Michigan				
Off Saugatuck	Lake Trout	50.8	ND	7

---

a ppt indicates parts per trillion which is equivalent to ng/kg.

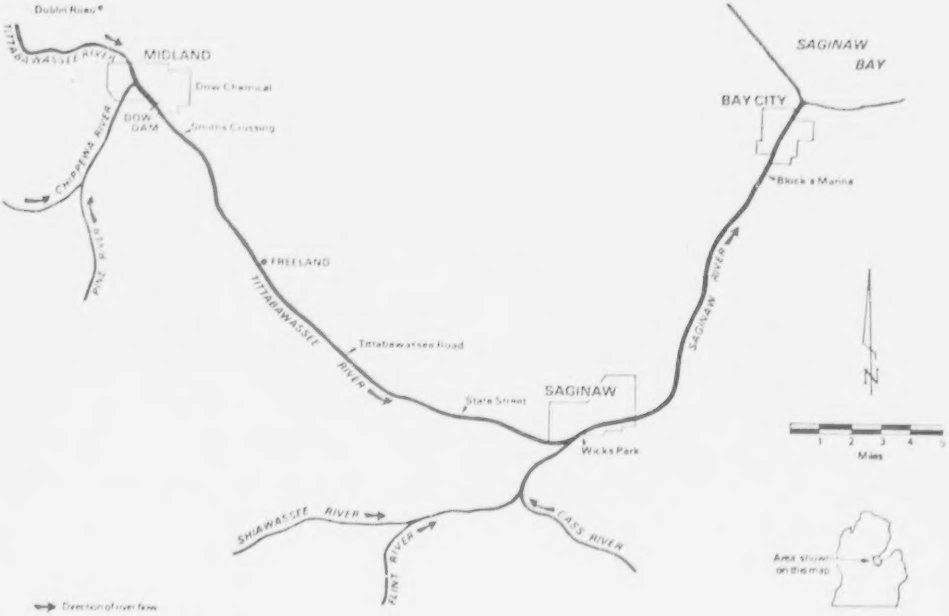
b signifies composite fish sample.

c ND indicates that TCDD was not detected in the fish sample at the corresponding detection limit.

* Skinless Fillets

FIGURE 1:

SAGINAW RIVER BASIN


Dublin Road*
TITTABAWASSEE RIVER
MIDLAND
Dow Chemical
DOW DAM
Smith's Crossing
CHIPPEWA RIVER
PINE RIVER
FREELAND
TITTABAWASSEE RIVER
Tittabawassee Road
State Street
SAGINAW
Wicks Park
SHIAWASSEE RIVER
FLINT RIVER
CASS RIVER
SAGINAW RIVER
BAY CITY
Brick's Marina
SAGINAW BAY
N
1
2
3
4
5
Miles
Area shown on this map

→ Direction of river flow

* Approximately 3 river miles upstream from confluence of Chippewa and Tittabawassee Rivers

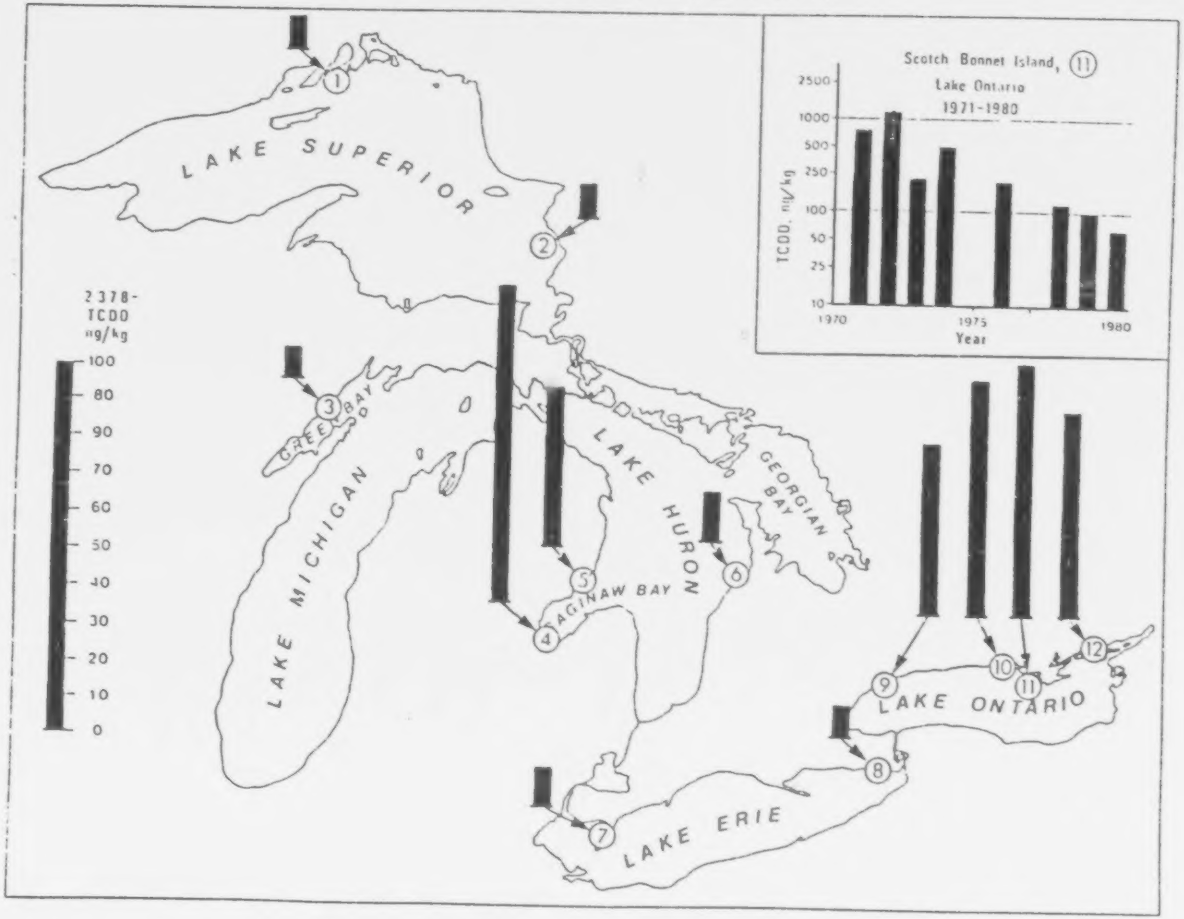
G. Canadian Study on Gull Eggs from the Great Lakes

Dr. Douglass Hallet of the Canadian Wildlife Service reported finding 2,3,7,8-TCDD in Herring gull eggs, collected in 1980, at 12 different locations within the Great Lakes (32). The highest levels of 2,3,7,8-TCDD were found in Saginaw Bay eggs (43-90 ppt). Levels in Lake Ontario eggs were slightly lower and ranged from 44-64 ppt. Lower levels (approximately 3-10 ppt) were found in eggs collected at other Great Lakes sites. The 2,3,7,8-TCDD isomer was found to predominate (>90%). These results are graphically presented in Figure 2. There have been no reports of abnormalities in gull chicks or mature birds. Egg samples collected from Lake Ontario in 1971 and preserved until analysis in 1980 had 2,3,7,8-TCDD levels of 800 ppt. It was conjectured that the decrease in 2,3,7,8-TCDD levels since 1971 (Figure 2) might be attributed to the discontinuation of 2,4,5-trichlorophenol production by Hooker Chemical at Niagara Falls, New York in 1971. Confirmational analyses of gull egg samples are now underway in U.S. laboratories.

H. Gull Flesh Analyses From the Great Lakes (December 1980)

Two flesh samples from gulls in the Saginaw Bay were recently analyzed by Dr. David Stalling of the Columbia National Fisheries Research Laboratory (30). In the two samples, the 2,3,7,8-TCDD isomer predominated (>90%) and was found at levels of 70 and 160 ppt (31). HxCDDs and OCDDs were also found resulting in a total PCDD level of 186 and 196 ppt. Total PCDFs were found at 117 and 121 ppt with the PnCDFs and HxCDFs being the predominate furans.

Figure 2: "Dioxin in Gull Eggs" (Reference 27)


LAKE SUPERIOR
LAKE MICHIGAN
LAKE HURON
GEORGIAN BAY
GREEN BAY
SAGINAW BAY
LAKE ERIE
LAKE ONTARIO
1
2
3
4
5
6
7
8
9
10
11
12
2378-
TCDD
ng/kg
100
80
90
70
60
50
40
30
20
10
0
Scotch Bonnet Island, ⑪
Lake Ontario
1971-1980
TCDD, ng/kg
2500
1000
500
250
100
50
25
10
1970
1975
1980
Year

I. PCDD and PCDF Analyses on Great Lakes Fish (April 1981)

1. The Columbia National Fisheries Research Laboratory has obtained positive results for TCDDs and TCDFs on composite whole fish samples from the Great Lakes (29). This fish sampling is being conducted under the National Pesticides Monitoring Program. Huron and Ontario fish samples ranged in TCDD concentrations from 33 to 94 ppt. The highest values were reported in fish collected from Saginaw Bay in 1978. Data collected to date is presented in Table 6. Tittabawassee fish data (see E. above) is included for comparison. The predominate (>90%) isomeric form of TCDD discovered has been 2,3,7,8-TCDD (30,31). The majority (>90%) of the other chlorinated dioxins and furans found in fish appear to have the 2,3,7,8 ring positions chlorinated (30,31). Therefore, the potential toxicity of these dioxins and furans may approach that of 2,3,7,8-TCDD (3). Very preliminary data indicates that the isomeric distribution of PCDDs and PCDFs in river sediments do not reflect those found in fish (31,33). Differences in solubilities of PCDDs and PCDFs, absorption or retention of these chemicals could account for the observed difference in distribution (33).

2. The FDA has also reported finding TCDDs in fish from Saginaw Bay. Analyses performed by FDA on edible fillets from 27 fish collected from Saginaw Bay in 1979, determined four to be positive for TCDDs (21 to 45 ppt). FDA recommended that fish having TCDD residues above 100 ppt should not be consumed and even lower levels, down to 50 ppt, consumption should be limited. FDA stated that no adverse health consequences would be anticipated from the moderate consumption of fish containing TCDD residues below 50 ppt (34).

TABLE 6

Residues of Dibenzofurans and Dibenzo-p-dioxins in Composite Samples of Fish and Gulls From Different Sites on the Great Lakes. Residues are in parts-per-trillion and by Number of Chlorine Substitutions (29).

Site and Sample	Dibenzofurans						Dibenzo-p-Dioxins						Sample Index No.
	4Cl	5Cl	6Cl	7Cl	8Cl	Total	4Cl	5Cl	6Cl	7Cl	8Cl	Total	
Saginaw Bay, L. Huron Carp	27	44	34	44	4	153	94	157	122	12	ND	385	GL1
Saginaw Bay, L. Huron Herring Gull #1	16	28	57	17	3	121	160	ND	20	ND	19	199	GL2
Saginaw Bay, L. Huron Herring Gull #2	15	50	40	7	5	117	70	ND	38	ND	28	186	GL3
Bay Port, L. Huron Carp	5	12	5	6	1	27	27	21	ND	31	32	111	GL7
L. Huron 79-06 (Duplicate analyses) Lake Trout (1)	32	27.5	16.3	8.7	6.3	91							GL13
(2)	19	29	16	4	2	70							
Tittabawassee River-below Dow Dam Carp	37	73	145	31	4	290	81	31	44	53	14	223	GL9
L. St. Clair 79-12 Walleye	4	4.2	0.7	0.7	2.6	12							GL14
L. Erie 79-09 Walleye	17.6	9.2	5.7	5.3	2.2	40							GL15
Port Clinton, L. Erie Carp	5	5	2	4	1.5	17.5	ND	9	ND	11	30	50	GL8
Saugatuck, L. Michigan Lake Trout	35	41	8	1.4	1.4	87	5	ND	ND	ND	ND	5	GL4

TABLE 6 (Continued)

Residues of Dibenzofurans and Dibenzo-p-dioxins in Composite Samples of Fish and Gulls From Different Sites on the Great Lakes. Residues are in parts-per-trillion and by Number of Chlorine Substitutions (29).

Site and Sample	Dibenzofurans						Dibenzo-p-Dioxins						Sample Index No.
	4Cl	5Cl	6Cl	7Cl	8Cl	Total	4Cl	5Cl	6Cl	7Cl	8Cl	Total	
L. Michigan-79-check Lake Trout	33	61	10.4	4.4	2	110							GL16
Keweenaw, L. Superior Bloater Chub	26	9	2	1	2	40	ND	ND	ND	ND	ND	ND	GL5
Isle Royale, L. Siskiwit, L. Superior Lake Trout	10	5	ND	ND	Trace	15	ND	ND	ND	ND	Trace	Trace	GL10
L. Superior 79-02 Lake Trout	19	8.8	5.4	3.7	2.7	40							GL12
Roosevelt Beach, L. Ontario, Brook Trout	19	4	3	3	3	32	33	ND	ND	ND	ND	33	GL6
L. Ontario 79-10 Lake Trout	34	48	29	6	2	119							GL11

3. The New York State Department of Health is currently screening Lake Ontario fish for contamination. Previous analyses conducted on two fish determined dioxin contamination at 4.6 and 6.5 ppt (35). A potential source of contamination may be from landfills in the Niagara River area where 2,4,5-trichlorophenol wastes were dumped (36). Sediment samples from streams which drain into the Niagara River have been reported to contain dioxin (37).

J. NIEHS Analyses of a Michigan Dairy Herd Tissue Samples and Michigan's Study on Pentachlorophenol Treated Wood (1978-1979)

Studies by NIEHS of a dairy herd in Michigan, presumed to be contaminated by contact with PCP treated wood, revealed fat and liver contamination, respectively, with OCDDs at 7 and 15 ppb; HpCDDs at 6 and 15 ppb; and HxCDDs at 1 and 9 ppb (6). The presence of these contaminants was suspected of causing the illnesses noted in the cattle. Analyses conducted for PCDDs, by the State of Michigan, on the PCP treated wood in barns established the link between the wood and the presence of PCDDs in the cattle samples (38). OCDDs ranged from 289-5000 ppb, HpCDDs from 9-1161 ppb, and HxCDDs from non-detectable to 3.7 ppb in the wood.

K. U.S. EPA Assessment of Environmental Contamination in the Area of Hemlock Michigan (October, 1980)

As a result of numerous health complaints involving humans and domestic animals, the U.S. EPA conducted an environmental assessment in 1980 of the Hemlock, Michigan area, located 14 miles south of Midland (39). Citizens were dissatisfied with previous health and environmental studies (negative findings) conducted by county and state agencies. Residents were concerned that potable groundwater had been contaminated from a brine reinjection system operated by Dow Chemical in that area. A study of TCDDs in 40 environmental and biological samples yielded only one positive result at a detection limit of low ppt levels. Several fish, downspout soils, house dusts, a well sediment sample, a septic tank sludge, and sediment samples from Dow's injection systems were found to be negative for TCDDs at detection levels ranging from 1-43 ppt (40). The one positive sample was a caged fish collected from the Tittabawassee River, downstream of Midland, which contained 23 ppt TCDDs.

L. Crete Metals, Crete, Illinois (December, 1980)

TCDDs and TCDFs analyses were performed in 1980 on environmental and biological samples as a result of health complaints and the death of several horses near a wire reclamation facility (41). In the reclamation process, insulating and other materials were burned off wires, x-ray plates and transformer cables and other unknown metal containing products. Samples analyzed and results obtained are presented below in Table 7.

TABLE 7

	TCDDs (ppt)	TCDFs (ppt)
Stack #2 (Scrapping)	410	11,600
Furnace #2 (Scrapping)	58	730
Soil (on site)	21	230
Adipose (dead horse)	45	165

A variety of isomeric forms of TCDDs and TCDFs were detected, however, the analytical technique utilized could not adequately discriminate and quantify the isomers.

V. Current Government Activity

Except for disposal requirements for chlorophenolic wastes containing 2,3,7,8-TCDD, no other regulations exist for controlling PCDDs and PCDFs release to the environment (42). The implications of these data on the consumption of dioxin contaminated fish are currently undergoing review by various government agencies in this country and Canada.

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Long-Term Hazards of Polychlorinated Dibenzodioxins and Polychlorinated Dibenzofurans[*]

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During January 10–11, 1978 in Lyon, France, a joint National Institute of Environmental Health Sciences/International Agency for Research on Cancer *ad hoc* Working Group considered and discussed the feasibility of coordinating epidemiological studies on the long-term hazards associated with the chlorinated dibenzo-*p*-dioxins and chlorinated dibenzofurans (PCDDs and PCDFs). Nineteen invited scientists from eight countries presented introductory working papers summarizing the most up-to-date and relevant information available from their individual programs. This report represents the collective views and scientific opinions of the Working Group. The greater part of this document comprises epidemiological studies related to episodes of human exposure. The review begins with a brief section concerning possible routes of human exposure, an overview of the pertinent chemical characteristics, and the salient toxicological properties of the structurally siimilar PCDDs/PCDFs. The Working Group report ends with recommendations for future activities.

Introduction

Human exposure in the workplace can occur when chlorinated dibenzo-*p*-dioxins (PCDDs) are formed during the production of certain compounds such as the herbicide 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), the fungicide pentachlorophenol, and the germicide hexachlorophene (*1*). The dioxins, impurities/contaminants associated with these end-products, result most often from treatment of chlorinated benzenes at elevated temperature and pressure under alkaline conditions. Via the widespread use of these commercial products (*1, 2*) the general population may also become exposed.

In recent years, out-of-control chemical reactions during the production of 2,4,5-trichlorophenol have proceeded to the explosive stage thereby exposing persons to toxic levels of PCDDs. From certain of these accidents, moreover, not only did those occupationally involved receive dangerous exposure but also those inhabiting the surrounding areas received uncommon risk.

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The structurally-related chlorinated dibenzofurans (PCDFs) are contaminants found in some polychlorinated biphenyl compounds: Aroclor, Clophen, Phenoclor (*3*).

These impurities are more toxic and represent a greater environmental hazard than the compounds they contaminate (*1, 3-9*). For instance, 2,3,7,8-tetra-CDD has been recently found in beef fat from cattle grazed on 2,4,5-T-treated rangeland and in breast milk from women living in areas where 2,4,5-T is used on rangeland or in forest areas (*10*). Various investigators (*11-14*) have also discovered PCDDs and PCDFs in fly ash and flue gas from municipal incinerators, and in dust from fungicide-treated wood (*15*).

Chemical Aspects

The PCDDs and PCDFs are two series of tricyclic aromatic compounds which exhibit similar chemical and physical properties (*1, 3, 4*). The basic two-dimensional structures (I, II) have eight possible points of chemical addition. From the monochloro to the octachloro derivatives, a variety of positional isomers are possible: 75 PCDDs and 135 PCDFs (Table 1).

The extreme toxic potency of some of these compounds, as well as the large number of potential isomers, warrant analytical methods exhibiting high sensitivity and specificity to monitor the environment. A desirable detection limit of one part per trillion (ppt, 1 picogram/gram sample) is being reached with current methodology. Essential requirements included efficient clean-up, good separation and selectivity, ultrasensitive quantification, and validation (*16, 17*).

Toxicological Aspects

Animals

The prototype and most extensively studied isomer of the PCDDs and PCDFs is the 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-tetra-CDD), perhaps the most potent man-made toxin presently known. The comparative oral lethal dose values for various PCDDs in mice and guinea pigs (Table 2) show clearly that the 2,3,7,8-tetra-CDD and the 1,2,3,7,8-penta-CDD isomers are the most toxic (*18*).

Toxic effects induced by PCDDs and PCDFs vary quantitatively and qualitatively among different species; however, within a single species the untoward consequences are markedly similar for all PCDDs and PCDFs that have been studied. For example, the toxic effects induced by 2,3,7,8-tetra-CDD which are most often observed in mice, guinea-pigs, and monkeys, are illustrated in Table 3 (*20*). In a brief report, daily oral intake (12-61 days) of <1 µg/kg body weight 2,3,7,8-tetra-CDD was stated as being lethal to young male rhesus monkeys (*21*), whereas McConnell et al. (*22*) reported an LD_{50} in female rhesus monkeys as <70 µg/kg body weight.

A preliminary report on 2,3,7,8-tetra-CDF concerning the toxicity of PCDFs (*23*), and a report on PCDFs (*24*) have been published.

The toxic syndrome produced by PCDDs and PCDFs may be divided into seven categories:

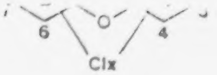
Chloracne. 2,3,7,8-Tetra-CDD and the tri-PCDF and tetra-PCDF were found to be active skin irritants and to induce acneform lesions in the skin of rabbit ears (*25*).

Classical chloracne is a hallmark of PCDD exposure in humans and an analogous hyperkeratosis and the modulation of sebaceous structures to keratin cysts have been observed in monkeys, rabbits, and hairless mice.

Chloracne or acneform dermatitis is a common occupational dermatitis characterized by comedones, keratin cysts, pustules, papules, and abscesses. In 1957, Kimmig and Schulz (*26*) found that 2,3,7,8-tetra-CDD was the agent responsible for causing occupational chloracne in employees of chlorophenol-producing factories. Further, in 1971 PCDDs were implicated as causing chloracne in male workers in a plant producing 2,4-D and 2,4,5-T (*27, 28*). Chloracne may appear weeks or months after the initial exposure to PCDDs and PCDFs.

Hepatoxicity. The degree of hepatic involvement appears to be dose-dependent, and the severity of the changes produced varies between species (*29*). Hepatic necrosis produced by 2,3,7,8-tetra-CDD is probably a contributing cause of death in rats and rabbits, while hepatic necrosis and liver insufficiency are less extensive in mice and are minimal in comparison in guinea pigs and monkeys (*22, 30-37*). Hepatic porphyrin accumulation has been observed in mice, rats, and chickens.

Hypoplasia of the Lymphoid Tissues. Particularly involved are the cortical cells of the thymus and this hypoplasia has been observed in mice, rats, guinea-pigs, and monkeys. The most significant findings in both mice and



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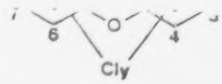

II

guinea-pigs treated with sublethal doses of 2,3,7,8-tetra-CDD were in the lymphoid system, resulting in suppression of cell-mediated immunity, particularly in young animals (*36, 38*). Low levels of 2,3,7,8-tetra-CDD that did not produce overt clinical or pathological changes still reduced host defences: 1 μg/kg bw given orally once weekly for 4 weeks to mice before infection with Salmonella increased mortality and decreased the time from infection to death (*39*). The increased mortality may be caused by the endotoxin content of these gram negative bacteria, since 2,3,7,8-tetra-CDD markedly increases the susceptibility of mice to endotoxin (lipopolysaccharide of *Escherichia coli*) (*40*). Treatment of female mice and rats with 2,3,7,8-tetra-CDD during the latter half of gestation and in the postnatal period resulted in a severe depletion of lymphocytes in the thymic cortex of the offspring (*41*). Cellular immunity was impaired.

Hematological changes in mice, rats, and guinea pigs treated with 2,3,7,8-tetra-CDD include lymphopenia and thrombocytopenia (*42, 43*); manifest also is an increased susceptibility to infection concomitant with the suppression of cell-mediated immunity. For 9 months, female rhesus monkeys received a diet containing 500 ppt 2,3,7,8-tetra-CDD; within 6 months, the monkeys became anemic and after 9 months pancytopenic (*44*). The marked thrombocytopenia was associated with widespread hemorrhage. Death occurred in five of the eight monkeys between months 7 and 12 of the experiment at total exposure levels of 2,3,7,8-tetra-CDD of 2-3 μg/kg body weight. At autopsy, in addition to extensive hemorrhage, there was a distinct hypocellularity of the bone marrow and lymph nodes. Hypertrophy, hyperplasia, and metaplasia of the epithelium in the bronchial tree, bile ducts, pancreatic ducts, salivary-gland ducts, and palpebral conjunctivae were observed. Squamous metaplasia and keratinization of the sebaceous glands and hair follicles were present in the skin. Death was attributed to complications from the severe pancytopenia (*44*).

Table 1. Possible number of isomers for polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans.

Chlorine atoms	PCDD isomers[a]	PCDF isomers[b]
1	2	4
2	10	16
3	14	28
4	22	38
5	14	28
6	10	16
7	2	4
8	1	1
Total	75	135

[a] Chlorinated dibenzo-dioxins: empirical formula $C_{12}H_{7-0}Cl_{1-8}O_2$; molecular weight ranges, 218–460.

[b] Chlorinated dibenzofurans: empirical formula, $C_{12}H_{7-0}Cl_{1-8}O$; molecular weight ranges, 202–444.

General Debilitation and Wasting. Animals that receive a toxic or lethal dose of PCDDs or PCDFs exhibit a chronic and progressive weight loss with parallel mobilization of peripheral fat, increased serum triglyceride levels, and development of a fatty liver. Death due to PCDDs or PCDFs intoxication is delayed, as exemplified by an elapsed time period of 6 to 8 weeks following administration of a lethal dose and eventual death (*18, 22, 44*).

Embryotoxicity and Teratogenicity of 2,3,7,8-tetra-CDD. In repeated or single doses of 2,3,7,8-tetra-CDD to mice, as little as 1-10 μg/kg cause increased frequencies of cleft palate and kidney abnormalities (*45-48*) (see Table 4). In rats, embryo-lethal effects occur under experimental conditions (*49, 50*), and kidney anomalies (*45*), intestinal hemorrhages, and general edema can be produced in the fetuses (*51*). Few follow-up studies of the effects of prenatal exposure on postnatal functions have been published. In mice, fetal kidney abnormalities caused by 2,3,7,8-tetra-CDD may progress into a hydronephrosis during the postnatal period (*52*).

Chick Edema Disease. Hydropericardium, ascites, subcutaneous edema, liver necrosis, and death were described in 1957 following the accidental administration of toxic fats in the feed of broiler chickens (ascites have been observed also in mice) (*1*). The toxic material was later identi-

Table 2. Estimated single oral LD50-30 values of certain polychlorinated dibenzo-*p*-dioxin isomers.[a]

Chlorine isomer	Guinea pigs µg/kg	Guinea pigs µmole/kg	Mice µg/kg	Mice µmole/kg
2,8	>300,000	>1,180	—	—
2,3,7	29,444	120.41	>3,000	>10
2,3,7,8	2	0.006	283.7	0.88
1,2,3,7,8	3.1	0.009	337.5	0.94
1,2,4,7,8	1,125	3.15	>6,000	>14
1,2,3,4,7,8	72.5	0.185	825	2.11
1,2,3,6,7,8	70–100[b]	0.178–0.255	1,250	3.19
1,2,3,7,8,9	60–100[b]	0.153–0.255	>1,440	>3.67
1,2,3,4,6,7,8	>600	>1.400	—	—
1-NO_2-3,7,8	>30,000	>90	—	—
1-NH_2-3,7,8	>30,000	>99	—	—
1-NO_2-2,3,7,8	47.5	0.129	>2,000	>5.4
1-NH_2-2,3,7,8	194.2	0.576	>4,800	>14.2

[a] Data from McConnell et al. (*18*). The LD_{50-30} was calculated by the Spearman-Karber method (*19*).

[b] Estimated range due to variability in replicates.

fied in commercial oleic and stearic acids produced from inedible tallow recovered from animal hides; trichlorophenols and pentachlorophenols had been used in the curing of the hides. The edema causative was termed toxic fat and more specifically chick edema factor and was characterized by x-ray crystallography as 1,2,3,7,8,9-hexa-CDD (*53*). 2,3,7,8-Tetra-CDD, hexa-CDD, and octa-CDD have been also identified in several commercial fatty acids (*54*). Daily doses of 10 or 100 µg hexa-CDD/kg bw, or of 1 or 10 µg 2,3,7,8-tetra-CDD/kg bw, produced a positive response in the chick edema bioassay; 0.5% octa-CDD in the diet had no effect (*35*). Similar edematous effects were observed in rats, pigs, dogs, and monkeys, but not in guinea pigs (*55*). Decreased serum albumin may be associated with the edema; edema has been also shown to occur in chicks dosed orally with 2,3,7,8-tetra-CDF (*56*).

Table 3. Summary of the toxic effects of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin.[a]

	Mice	Guinea pigs	Monkeys (female)
Thymus involution	+++	+++	+++
Spleen reduction (white pulp)	+	+	+
Bone marrow hypoplasia	±	++	+
Liver megalocytosis/generation	+++	−	−
Bile duct hyperplasia	±	±	+++
Testicular degeneration	++	+++	NA
Renal pelvis hyperplasia	−	++	+
Urinary bladder hyperplasia	−	++	−
Adrenal cortical atrophy (*zona glomerulosa*)	−	++	−
Hemorrhage			
Intestinal	+	+	−
Adrenal	−	++	−
Ascites	++	−	+
Cutaneous lesions	−	−	+++

[a] Data from Moore (*20*).

Other Effects. In one or more species of laboratory animals, bone marrow hypoplasia, testicular degeneration, renal pelvis and urinary bladder hyperplasia, and hemorrhage in the intestines and adrenals have been observed.

Enzyme Induction. 2,3,7,8-Tetra-CDD and other halogenated dibenzo-*p*-dioxins and dibenzofurans stimulate a number of enzyme activities, most notably in the liver (*1*). 2,3,7,8-Tetra-CDD is a potent inducer of hepatic and renal microsomal drug metabolizing enzymes (*57-69*). Intoxication with 2,3,7,8-tetra-CDD results in a marked increase in the cellular smooth endoplasmic reticulum content of hepatic and renal cells (*60,69*). 2,3,7,8-Tetra-CDD can simultaneously activate and suppress certain microsome-associated foreign-compound and steroid-hormone-metabolizing enzyme systems (*63*) as well as increase the activity of both renal and hepatic glutathione-S transferase (*70*).

2,3,7,8-Tetra-CDD is the most active of the PCDDs in inducing hepatic δ-aminolevulinic acid (ALA) synthetase and aryl hydrocarbon hydroxylase (AHH) in chick embryo liver preparations (*71, 72*).

The PCDDs that induce ALA synthetase in chick embryo have two common properties: (1) halogen atoms occupy at least three of the four ring positions (2,3,7,8), and (2) at least one free, nonhalogenated carbon atom is unoccupied (*71*). The available toxicological data (*35*) indicate that those PCDDs that are lethal at low doses, teratogenic, or produce acne also induce ALA synthe-

Table 4. Evaluation of reported embryotoxic and teratogenic effects induced by 2,3,7,8-tetrachlorodibenzo-*p*-dioxins in rats and mice[a]

Species	Strain	Embryotoxic/ teratogenic effect	Dose, μg/kg		Period of dosing, days	Route	Reference
			Lowest Tested[b]	ED_{50}[c]			
Rat		Intestinal hemorrhage	0.125	0.5?		Oral	(*50*)
	CD	Kidney abnormality	0.5	>1	6–15	S.C.	(*45*)
Mouse	CD-1	Cleft palate	1?				
			3	>3	6–15	S.C.	(*45*)
		Kidney abnormality	1	1–3	6–15	S.C.	(*45*)
	DBA/2J	Cleft palate	3	>3	6–15	S.C.	(*45*)
		Kidney abnormality	3	>3	6–15	S.C.	(*45*)
	C57BL/6J	Cleft palate	3	>3	6–15	S.C.	(*45*)
		Kidney abnormality	3	<3	6–15	S.C.	(*45*)
	NMRI	Cleft palate	3	6.5	6–15	Oral	(*46*)
			9	<9	9–13	Oral	(*46*)
			15	40	13	Oral	(*47*)
			5	15	11	Oral	(*47*)

[a] Data from Neubert et al. (*47*).

[b] The lowest dose with which an embryotoxic or teratogenic effect detectable at birth has been produced is indicated. Since sometimes only one dose level was tested, this does not necessarily represent the lowest dose from which an effect could result.

[c] ED_{50}-dose required to produce an embryotoxic effect in 50% of animals.

tase; those PCDDs that are not toxic generally do not induce ALA synthetase. The structure-activity relation of PCDDs to induce AHH in chick embryos was identical to that in inducing ALA synthetase (*71*).

Mixed-function oxidase enzyme systems of mouse strains "non-responsive" to other aromatic hydrocarbons were induced by single doses of 2,3,7,8-tetra-CDD, as evidenced by increases in hepatic monooxygenase activities and in concentrations of cytochrome P-448 (*73-76*). Genetic resistance to induction of AHH by 3-methylcholanthrene in DBA/2J mice was overcome by treatment with 2,3,7,8-tetra-CDD. This result conflicts with the hypothesis that induction of AHH activity is a consequence of the formation of cytochrome P-448 (*77*). A component that has a high binding affinity for 2,3,7,8-tetra-CDD was found in mouse liver cytosol (*78*).

2,3,7,8-Tetra-CDD induces AHH even in 'poorly responsive' strains of mice (*79*), not only in liver but also in lung, kidney, and colon. The DBA/2N strain, which responds only weakly to the sarcomatogenic action of 3-methylcholanthrene, becomes highly susceptible after treatment with 2,3,7,8-tetra-CDD (*74, 80*).

2,3,7,8-Tetra-CDD is approximately 30,000 times more potent than 3-methylcholanthrene in inducing AHH activity in rat liver (*81*).

McConnell et al. (*18*) reported that the comparative toxicity of 13 PCDDs in mice and guinea-pigs (see Table 2) supports the idea that the relative potency (or rank order) of a congener to produce one toxic response is a good indicator of its relative potency (or rank order) to produce other toxic manifestations.

Hepatic Cytosol Binding Protein. A macromolecular binding species has been characterized in the hepatic cytosol fraction of mouse and rat liver which has the *in vitro* binding properties predicted for the receptor for the induction of AHH activity based on the *in vivo* biology (*78*): namely, (1) ^{3}H-2,3,7,8-tetra-CDD binds to this cytosol protein reversibly with a high affinity (K_d = 0.27 *nM*) comparable to the ED_{50} for hepatic AHH induction (ED_{50} in mice = 1 nmole/kg); (2) the binding affinity of halogenated dibenzo-*p*-dioxins and dibenzofurans for this protein *in vitro* corresponds to their potency to induce hepatic AHH activity in the chicken embryo; and (3) other compounds, such as the polycyclic aromatic hydrocarbons, which induce AHH activity and cytochrome P_1-450 also compete for this cytosolic binding protein, but compounds which induce other types of microsomal monooxygenase activities (e.g., phenobarbital) and steroids fail to bind. Thus, this cytosolic binding protein may be the receptor for the induction of AHH activity.

Structure-Activity Relations. The pathologic effects produced by the toxic PCDD and PCDF isomers are similar to those of 2,3,7,8-tetra-CDD for a given species, differing only in the intensity of the toxic effect produced by a given isomer. The toxic PCDDs have chlorine atoms in at least three of the four lateral ring positions (2,3,7, and 8) with at least one unsubstituted ring position (the octa-CDD is comparatively inactive). To the extent that the toxicity has been determined, a similar structure-toxicity relation has been observed for the PCDFs.

The structure-activity relation established for PCDDs and PCDFs for the induction of hepatic aryl hydrocarbon hydroxylase (AHH) activity and for binding to the hepatic cytosol binding species has been extended to other classes of chlorinated aromatic compounds. 3,4,3′4′-Tetrachloroazoxybenzene (TCAOB) and 3,4,3′,4′-tetrachloroazobenzene (TCAB) are potent acnegens formed as trace contaminants in the synthesis of 3,4-dichloroaniline or herbicides based on this compound (*82*). At high doses in animals, TCAB is reported to produce thymic involution and liver damage similar to 2,3,7,8-tetra-CDD (*83*). Both TCAOB and TCAB are potent inducers of hepatic AHH activity and bind to the hepatic cytosol binding protein with a high affinity. Congeners such as 3,5,3′5′-tetrachloroazoxybenzene and 3,5,3′,5′-tetrachloroazobenzene fail to induce AHH activity, fail to bind to the hepatic cytosol species, and fail to produce chloracne.

Of 16 halogenated biphenyl compounds tested, only 3,4,3′4′-tetrachloro-, 3,4,5,3′4′5′-hexachloro- and 3,4,5,3′4′5′-hexabromobiphenyls induced hepatic AHH activity and bound to the hepatic cytosol binding species (*84*). The 3,4,3′4′-tetrachlorobiphenyl has been reported to produce chloracne. McKinney et al. (*56*) found that of five hexachlorobiphenyls tested in chickens, 3,4,5,3′4′5′-hexachlorobiphenyl was by far the most toxic, and the only one that induced significant chick edema and involution of the thymus.

Pharmacokinetics. In the rat, following acute or chronic administration, 2,3,7,8-tetra-CDD is accumulated primarily in the liver and to a lesser extent in the fat, and is largely eliminated unmetabolized in the feces with a whole body half-life of about 3 weeks. Some pharmacokinetic experiments suggest the formation of a polar metabolite appearing in the urine, but direct attempts to demonstrate metabolism with hepatic microsomes *in vitro* have been negative (*1*).

Mutagenicity. Only four dioxin isomers have been evaluated for mutagenicity: the 2,7-di-, 2,3,7,8-tetra, and octa-CDDs as well as the unsubstituted dibenzo-*p*-dioxin (*85*).

2,3,7,8-Tetra-CDD increased the reversion frequency to streptomycin independence in *Escherichia coli* Sd-4. In *Salmonella typhimurium*, frameshift mutations in strain TA1532, but not base substitutions in strain TA1530, were induced by toxic concentrations of 2,3,7,8-tetra-CDD (*86*). In plate assays, the response was positive with *S. typhimurium* TA1532, doubtful with TA1531 and TA1534, and negative with G46 and TA1530 (*87*).

McCann (*88*) tested 2,3,7,8-tetra-CDD in *S. typhimurium*, both with and without metabolic activation, using a spot-test and the standard plate test with strains TA1532, TA1535, TA1537, and TA1538; all these tests were negative.

Octa-CDD was nonmutagenic in *S. typhimurium* strains G46, TA1530, and TA1531, and doubtful results were obtained with strains TA1532 and TA1534 (*87*). Metabolic activation systems were not included in any of these microbiological assays.

Inhibition of mitosis and chromosomal abnormalities (dicentric bridges and chromatin fusion with formation of multinuclei or a single large nucleus) were observed in endosperm cells of the African blood lily (*Haemanthus Katherinae* Baker) treated with 2,3,7,8-tetra-CDD in the presence or absence of 2,4,5-T (*89*).

No chromosomal aberrations were observed in bone marrow cells of male rats treated with 2,7-di-CDD, 2,3,7,8-tetra-CDD, or dibenzo-*p*-dioxin by oral intubation, with 2,3,7,8-tetra-CDD by intraperitoneal injection or orally (*90*). However, when Osborne Mendel rats of both sexes were treated twice weekly for 13 weeks with 2,3,7,8-tetra-CDD, a significant but weak increase in the number of chromosome aberrations in bone marrow cells was reported (*91*).

2,3,7,8-Tetra-CDD did not induce dominant lethal mutations in Wistar rats after oral administration to males for 7 days (*51*).

Carcinogenicity. Two reports indicate that chronic administration of low levels of 2,3,7,8-tetra-CDD to rats is associated with an increased incidence of neoplasia (*1, 92, 93*).

Groups of 10 male Sprague-Dawley rats were fed a diet containing 2,3,7,8-tetra-CDD for 78 weeks in the following amounts (figures in parentheses are approximate weekly doses): 0, 1 ppt (0.0003 μg/kg body weight) 5 ppt (0.001 μg/kg), 50 ppt (0.01 μg/kg), 500 ppt (0.1 μg/kg), 1 ppb (0.4 μg/kg), 5 ppb (2.0 μg/kg), 50 ppb (24 μg/kg), 500 ppb (240 μg/kg), and 1000 ppb (500 μg/kg). The three highest dose levels (50, 500, and 1000 ppb) were toxic and killed all animals by the fourth week. Of the six remaining test groups, the overall incidence of neoplasms was 23/60 (38%); none occurred in the 1 ppt group. In the 5 ppt group, 5/10 animals had 6 neoplasms [ear-duct carcinoma, lymphocytic leukemia, adenocarcinoma, malignant histocytoma (with metastases), angiosarcoma, Leydig-cell adenoma]; the following groups also showed neoplasms: 50 ppt, three observed in 3/10; 500 ppt, four 4/10; 1 ppb, five observed in 4/10; 5 ppb, ten observed in 7/10. Neoplasms were not observed in the controls (*92*).

Groups of 100 Sprague-Dawley rats (50 males and 50 females) received for two years diets containing 0, 22, 210, and 22,000 ppt, equivalent to 0.0, 0.001, 0.01, and 0.1 µg 2,3,7,8-tetra-CDD/kg/day. Continuous ingestion of 0.001 µg/kg/day did not cause any chemically related changes in tumor incidence or toxicity; feeding with 0.01 µg/kg/day induced an increased incidence ($p < 0.05$) of hepatocellular hyperplastic nodules (female: 18/50 versus 8/86 controls), of focal alveolar hyperplasia in the lungs, and of urinary excretion of porphyrins (female). Dietary intake of 0.1 µg/kg/day caused an increased incidence ($p < 0.05$) of hepatocellular carcinomas (female: 11/49 versus 1/86) and squamous-cell carcinomas of the lung (female: 7/49 versus 0/86), of the hard palate/nasal turbinates (male: 4/50 versus 0/85; female: 4/49 versus 0/86), and of the tongue (male: 3/50 versus 0/85). Further increased were adenoma of the adrenal cortex (male) and hepatocellular hyperplastic nodules (female). At this dose, certain age-related lesions were reduced (males: acinar adenoma of the pancreas; females: granulosal cell neoplasm of the ovary, benign and malignant tumors of the mammary gland, pituitary adenoma, and benign tumors of the uterus). Also, chronic administration of 2,3,7,8-tetra-CDD caused multiple toxicologic effects, including increased mortality, decreased body weight gain, slight depression of certain hematologic parameters, increased urinary excretion of porphyrins and δ-aminolevulinic acid, increased serum levels of alkaline phosphatase, glutamyl transferase and serum glutamic pyruvic transaminase, and morphologic changes primarily of the hepatic, lymphoid, respiratory, and vascular tissues of the body (*93*).

These two reports show that chronic administration of 2,3,7,8-tetra CDD causes an increased incidence of neoplasms, but not whether 2,3,7,8-tetra-CDD acts as an initiator or promoter. This consideration is particularly important because unequivocal evidence is lacking that 2,3,7,8-tetra-CDD is a mutagen or is metabolized, and no evidence is available that 2,3,7,8-tetra-CDD and/or metabolite(s) bind covalently to macromolecules.

As summarized in Table 5, at least 24 long-term carcinogenicity studies on mice and rats are in progress (*94*).

Humans

Toxic Effects in Humans. Toxicity due to 2,3,7,8-tetra-CDD has been reported after occupational exposure during the industrial synthesis of 2,4,5-trichlorophenol (TCP) and 2,4,5-T, after exposure in factories and in the surrounding environment due to accidents occurring during the synthesis of TCP, and after exposure to herbicides and other materials containing 2,3,7,8-tetra-CDD. Exposed subjects have been found to develop a wide variety of lesions and symptoms (Table 6). For instance, a typical exposure victim experiences a number and a variety of clinical signs and symptoms: early exposure symptoms may include a burning sensation of the eyes, nose, and throat followed by headache, dizziness, nausea, and vomiting. Some days later, severe itching, redness, swelling of the face, more marked over the eyelids, nose and lips, may develop. Within the initial weeks after exposure, inflamed nodules as well as pustules appear on the face, forearms, shoulders, neck, and trunk, leading then to comedones and cysts. After a month or two, acneform eruptions emerge and the skin becomes hyperpigmented. At about the same time, aching muscles, mainly in the thighs and chest area, become manifest and aggravated on exertion. Insomnia, extreme irritability, and loss of libido also occur during this stage.

Other than the consistently found clinical feature of acne, other findings in humans may include: neuromuscular symptoms (weakness and pain with nerve conduction abnormalities), porphyria cutanea tarda, hepatic dysfunctions, hyperlipidemia, cutaneous hyperpigmentation and hirsutism, chronic eye irritation, emotional disorders, and neuropsychiatric syndromes.

Chloracne, one of the most constant and prominent features of 2,3,7,8-tetra-CDD exposure, has been described as a refractory acne characterized by inclusion cysts, comedones and pustules, with eventual scarring of the skin, more frequently originating on the face and sometimes spreading to other parts of the body. Many patients also have blepharoconjunctivitis and irritation of other mucous membranes. Sometimes the chloracne is preceded by erythematous and edematous skin lesions. The latent period between exposure and the appearance of clear signs of chloracne ranges from a few weeks to several months (*106*).

An important and unique episode revolves around three scientists who were self-exposed to 2,3,7,8-tetra-CDD: one heated trichlorophenol in an alkaline solution, a second heated potassium trichlorophenate, and a third worked in the same laboratory as the second and used a diluted solution of the synthesized dioxin (*107*). The first two scientists developed chloracne eight weeks after exposure, whereas the third showed no evidence of the characteristic acneform lesions. Delayed symptoms, probably due to 2,3,7,8-tetra-CDD, developed approximately two years later, and the second two scientists showed personality changes

Table 5. Ongoing long-term carcinogenicity testing of chlorinated dibenzo-*p*-dioxins.[a,b]

Compound	Number of studies	Route	Species
Dibenzo-*p*-dioxin	4	Oral (diet)	Mouse, rat
		Skin	Mouse
			Mouse (with DMBA)[c]
2,7-Di-CDD	4	Oral (diet)	Mouse, rat
		Skin	Mouse
			Mouse (with DMBA)[c]
2,3,7-Tri-CDD	3	Oral (gavage)	Mouse, rat
		Skin	Mouse
2,3,7,8-Tetra-CDD[d]	5	Oral (diet)	Rat
		Oral (gavage)	Mouse
		Oral (gavage)	Mouse, rat
		Skin	Mouse
1,2,3,6,7,8-Hexa-CDD[d]	3	Oral (gavage)	Mouse, rat
		Skin	Mouse
1,2,3,7,8,9-Hexa-CDD[d]	2	Oral (gavage)	Mouse
		Skin	Mouse (with 1,2,3,6,7,8-hexa-CDD)
1,2,3,4,6,7,8,9-Octa-CDD	3	Oral (diet)	Mouse, rat
		Skin	Mouse

[a] Data taken from Information Bulletin on the Survey of Chemicals Being Tested for Carcinogenicity, No. 7 (*94*). Details of these studies as well as the authors/institutes are available in this source reference document.

[b] As of October 1979, carcinogenesis bioassay testing within the National Toxicology Program included unsubstituted dibenzo-*p*-dioxin (UDD; CAS 262-12-4; NCI Tech. Rept. No. 122), considered not carcinogenic to Osborne-Mendel rats and B6C3F1 mice when administered in feed at dose levels of 5 and 10 ppb, and 2,7-dichlorodibenzo-*p*-dioxin (DCDD; CAS 22857-26-0; NCI Tech. Rept. No. 123), considered not carcinogenic to Osborne-Mendel rats and female B6C3F1 mice but suggestive of a carcinogenic effect in male B6C3F1 male mice when fed concentrations of 5 and 10 ppb. Studies are in progress on 1,2,3,6,7,8-hexachlorodibenzo-*p*-dioxin (HCDD, CAS 57653-85-7) and 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD: CAS 1746-01-6) by gavage to Osborne-Mendel rats and B6C3F1 mice and by skin painting on Swiss mice. Testing on 2,3,7,8-tetrachlorodibenzofuran (TCDF; CAS 51207-31-9) is to begin in fiscal year 1980. For chemical disposition studies octachlorodibenzo-*p*-dioxin, 2,3,7,8-tetrachlorodibenzofuran, and the stereochemically related 2,4,3′,4′-tetrachloroazobenzene are also proposed.

[c] DMBA = dimethylbenzanthracene.

[d] The National Toxicology Program has completed and reported the results for the long-term carcinogenesis bioassay studies on 2,3,7,8-tetrachlorodibenzo-p-dioxin and 1,2,3,6,7,8-/1,2,3,7,8,9-hexachlorodibenzo-p-dioxins, with the following results.

2,3,7,8-tetrachlorodibenzo-p-dioxin—gavage, carcinogenic for Osborne-Mendel rats (increased incidences of follicular-cell thyroid tumors in males and of liver tumors in females) and carcinogenic for B6C3F1 mice (liver tumors in both sexes and thyroid tumors in females); dermal, carcinogenic for female Swiss-Webster mice (fibrosarcoma in the integumentary system) and although not shown carcinogenic for male Swiss-Webster mice, an increase in the same tumor type was observed. Mixture of hexachlorodibenzo-p-dioxins—gavage, carcinogenic for female Osborne-Mendel rats (hepatocellular carcinomas or neoplastic nodules), carcinogenic for male and female B6C3F1 mice (hepatocellular carcinomas and adenomas), and not demonstrated as carcinogenic for male Osborne-Mendel rats; dermal—not considered carcinogenic for male and female Swiss-Webster mice.

(mainly loss of energy and drive); impairment of vision, taste, and muscular coordination; sleep disturbances; gastrointestinal symptoms; and hirsutism. The first of the three experienced none of these adverse effects. All three exhibited hypercholesterolemia (>300 mg/100 ml).

Human Exposure to PCDDs and PCDFs

Major sources of human exposure to PCDDs and PCDFs include: exposure in the workplace; exposure in factories and in the surrounding environment from industrial accidents; exposure to contaminated materials, wastes, or food in the general environment; and exposure in Vietnam and other intensive herbicide spraying operations. (Prior and subsequent to use as a defoliant, 2,4,5-T was used in weed-killing and forest-thinning operations in the United States of America and elsewhere).

Occupational exposure may occur in manufacturing plants producing chlorinated phenols (tri-, tetra-, and pentachlorophenols), or phenoxy acid herbicides (2,4-D, 2,4,5-T), or PCBs; in factories utilizing these chemicals for the production of other substances (hexachlorophene from 2,4,5-trichlorophenol); in factories manufacturing or repairing transformers and capacitors or having heat exchange or heat hydraulic systems containing PCBs; and in the use processes of these chemicals under various occupational conditions such as spraying of herbicides, using chlorinated phenols for a variety of applications (especially wood preservative), sawing, or otherwise processing treated wood, and using hexachlorophene in sanitary occupations.

Table 6. Toxic effects of 2,3,7,8-tetrachlorodibenzo-p-dioxins in humans.

Effects	References
Dermatological	
Chloracne	(*25, 28, 95-108*)
Porphyria cutanea tarda	(*27, 104, 105, 109*)
Hyperpigmentation and hirsutism	(*27, 28, 107*)
Internal	
Liver damage[a]	(*27, 95, 98, 101, 102, 104-106, 110*)
Elevated serum hepatic enzyme levels	(*27, 28, 96, 104-106, 110*)
Disorders of fat metabolism	(*28, 104, 107*)
Disorders of carbohydrate metabolism	(*28, 101, 102, 104, 105*)
Cardiovascular disorders	(*101, 102, 104, 105*)
Urinary tract disorders	(*97, 101, 102*)
Respiratory disorders	(*95, 101, 102*)
Pancreatic disorders	(*101, 102*)
Neurological	
Polyneuropathies (peripheral neuritis)	(*101, 102, 104, 105*)
Lower extremity weakness	(*28, 95, 99, 101, 102, 104, 105, 108*)
Sensory impairments (sight, hearing, smell, taste)	(*28, 101, 102, 107, 110*)
Psychiatric	
Neurasthenic or depressive syndromes	(*28, 95, 99, 101, 102, 104, 105, 107*)

[a] Mild fibrosis, fatty changes, hemofuscin deposition and parenchymal-cell degeneration were observed in a few cases.

The burning of materials impregnated with commercial 2,3,4,6-tetrachlorophenates yielded 150-1000 μg of mixed PCDDs/g chlorophenate. Although only found as a minor constituent, 2,3,7,8-tetra-CDD has been quantified at levels exceeding 10 μg/g chlorophenate (*111*).

Pyrolysis of a technical grade PCB mixture yielded many PCDF isomers; the total yield could be as high as 3-25%. One of the main constituents is 2,3,7,8-tetra-CDF, the most toxic PCDF-isomer. Consequently, uncontrolled burning of PCBs can be an important environmental source of the hazardous PCDFs, and operations such as welding or soldering electrical equipment containing PCBs, or using casting waxes in foundries, may possibly entail a significant exposure (*112*). A PCB used in a heat exchange system for two years contained approximately 1.25 ppm of 2,3,7,8-tetra-CDF and a total of 15 ppm of PCDFs (*113*).

Apart from accidents such as the one which occurred in Seveso in 1976, general environmental exposure may originate from herbicide spraying and waste disposal.

An outbreak of PCDDs poisoning in humans, horses, and other animals occurred in Missouri in 1971 (*97, 114*) following the spraying of contaminated oil for dust control in horse arenas.

Another possible source of exposure to PCDDs and PCDFs pollution are waste oils, and possibly other waste, when burned both in industrial and municipal incinerators. Under simulated environmental conditions, the combustion of a standard 2,4,5-T formulation led to formation of small amounts of PCDDs and PCDFs (*115*). Buser and Bosshardt (*11*) quantified the total amount of PCDDs and PCDFs in fly ash from an industrial incinerator heating facility as 0.2 and 0.1 ppm, and in the fly ash from an industrial heating facility in Switzerland as 0.6 and 0.3 ppm. More than 30 individual PCDDs can be identified in the fly ash, but the known highly toxic PCDD isomers are only minor constituents (*12*). The number of PCDF isomers was larger; but, in this case, the known highly toxic isomers are major constituents (*13*).

An additional potential source of human exposure has been revealed: beef fat taken from cattle grazed on 2,4,5-T-treated rangeland contained 2,3,7,8-tetra-CDD; of the 11/14 positive samples, the four with the highest levels had 12, 20, 24, and 70 ppt (*10*). In another study, 3 of 24 samples of beef fat contained 3-4 ppt 2,3,7,8-tetra-CDD (*116*). Moreover, in a preliminary report of an ongoing enlarged study of women living in areas where 2,4,5-T is used on rangeland, 4/18 breast milk samples each contained approximately 1 ppt (*10*).

Workers in wood processing industries are exposed to wood dust containing preservatives as well as accompanying impurities and degradation products. Wood dust from a saw-mill in which a 2,3,4,6-tetrachlorophenol formulation was used as a fungicide was found to contain 1-10 ppm PCDFs and <0.5 ppm PCDDs (*15*).

In 1968, more than 1200 persons in South-west Japan were intoxicated by consuming a commercial rice oil contaminated with 1000 ppm PCBs. Nagayama et al. (*117*) analyzed the rice oil (Yusho oil) and found 5 ppm PCDFs, the major constituents of which were tetra- and penta-CDFs. Buser et al. (*113*) recently showed that 2,3,7,8-tetra-CDF was the main PCDF in the Yusho oil (0.45 ppm). The high level of PCDFs was caused by leakage from heat exchangers containing PCBs contaminated with PCDFs.

Workers manufacturing 2,4,5-trichlorophenol (TCP) or 2,4,5-T during normal production operations, and/or following explosions taking place in these plants, may have been exposed to a variety of polychlorinated chemicals whose type and quantity depends on the particular chemical processes in use and on the phase of the reaction in which the accident took place. Apparently, for instance, PCDFs are produced in the earlier stages when mainly tetrachlorobenzene is present and PCDDs are produced nearer the end of the reaction when primarily trichlorophenol is extant. The concentration of chlorophenols and chlorobenzenes, for example, was probably higher in the 1963 episode in The Netherlands, where the accident took place at the beginning of the reaction, then in the 1968 episode in the United Kingdom, or the 1976 accident in Italy, where the explosion took place at the end.

A thorough description of the reported industrial accidents and other cases of intoxications observed in exposed workers is given in the IARC Monograph (*1*). In this present report, only those episodes specifically discussed at length by the NIEHS/IARC *ad hoc* Working Group will be considered. (A sequential perspective of known accidents is given in Table 7).

In 1949, the first reported cases of industrial poisoning due to the formation of 2,3,7,8-tetra-CDD in uncontrolled exothermic reactions occurred during the manufacture of 2,4,5-trichlorophenol at a 2,4,5-T-producing factory in Nitro, West Virginia; 288 persons were affected (*99, 118*). In November 1953, an accident occurred in Ludwigshafen, Federal Republic of Germany, during the manufacture of TCP (*101,102,119,120*); 53 workers were affected by chloracne.

Five cases of chloracne were reported following an industrial accident in an Italian TCP-producing factory (*121*). In 1963, an accident occurred at the 2,4,5-T-producing factory in The Netherlands; approximately 50 persons were affected by chloracne (*122, 123*). In 1966, Dugois et al. (*98*) observed 21 cases of chloracne after an accident in a French factory producing TCP in the Grenoble region. In 1968, an accident occurred at the TCP-producing factory at Bolsover, Derbyshire, United Kingdom (*103, 106*). Within the next 7 months, 79 workers developed chloracne. In 1971, 2,3,7,8-tetra-CDD contaminated waste oil caused an outbreak of poisoning in humans, horses, and other animals (*97, 124*). In July 1976, an accident at the TCP-producing factory in Meda, Italy (*96*) resulted in the contamination of a large, densely populated area, including the towns of Seveso, Meda, Cesano Maderno, and Desio.

Key information concerning the effects of human exposure to herbicides in Vietnam, especially to the so-called "Agent Orange" (a 50:50 mixture of the *n*-butyl esters of 2,4-D and 2,4,5-T, containing up to 30 mg/kg or more 2,3,7,8-tetra-CDD), may be found in the literature (*1, 110, 125-127*).*

Exposure Episodes Considered in Detail by the NIEHS/IARC *ad hoc* Working Group

The text below is a condensed version of the discussions which took place during the two-day NIEHS/IARC *ad hoc* Working Group Meeting.

Phenoxy Acids Exposure

Cohort studies on herbicide sprayers have been conducted or are being planned in Scandinavian countries. The more advanced of these concerns Swedish railroad workers with exposure to a variety of herbicides. These people exhibited a significantly increased tumor incidence (apparently dose-dependent) and tumor mortality (*129*). The excess of tumors was found particularly among workers with exposure to amitrole (aminotriazole), whereas those exposed to phenoxy acids (2,4-D and 2,4,5-T) showed only a slightly increased excess of cancers. The study was small-sized, comprising 2978 person-years at observation in the total cohort and with 18 deaths *versus* 20.54 expected. The original conclusion from this study was that amitrole exposure may have caused an excess of tumors, whereas there was probably no pertinent increase in tumor incidence associated with exposure to phenoxy acids. The study has been recently reanalysed using a case-control approach, and through stratification on amitrole when considering the effect from phenoxy acids, and *vice versa*. The results show a possible and previously masked tumor-inducing effect also from phenoxy acids.

Another retrospective cohort mortality study, conducted in Finland on workers of five companies involved in spraying 2,4-D and 2,4,5-T on brushwood, did not show any increase in mortality. During 1955-1971, in the younger group of workers (under 45 years of age), however, four cancer deaths were observed versus less than two expected (no statistically significant difference). A prospective follow-up study for the period 1972-1976 revealed fewer cancer-related deaths than expected in all age groups. Clinical and anamnestic investigations were also performed showing a picture of acute complaints during and following the spraying operations: headache, transient dizziness, fatigue, abdominal complaints, skin and mucous irritations, and a few cases of persistent papulae.

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* More than 2,000,000 gallons of Agent Orange remaining from military defoliant operations were destroyed by incineration on board ship in the Pacific Ocean, 120 miles from Johnston Island. Temperatures not lower than 1250°C were used. The steel containers were melted (*128*).

Interestingly, some samples of 2,4,5-T used in Finland for spraying operations contained from 0.04 to 0.07 ppm 2,3,7,8-tetra-CDD. This may imply that the 2,4,5-T produced before 1965 contained lower levels of 2,3,7,8-tetra-CDD impurities than has been observed subsequently from 2,4,5-T used elsewhere. However, the analytical methods used in the determination of 2,3,7,8-tetra-CDD in the Finnish samples were antiquated in view of the rapid advancements made in analytical methodology and thus a confirmatory analysis of the samples using modern techniques was made by Rappe et al. (*130*). Five samples of 2,4,5-T ester dating from 1962 to 1967 were analysed for PCDDs and PCDFs; levels of 0.1-0.95 ppm 2,3,7,8-tetra-CDD and 0.1-0.15 ppm tetra-CDF were found.

A second feasibility study was designed and conducted in Finland to determine whether one could obtain anamnestic, clinical, hematological, and immunological information on Finnish railway and forestry workers who had been exposed for several years to herbicides containing 2,3,7,8-tetra-CDD. The exposed group consisted of 30 men who were control-matched to persons of the same age coming from the same district with similar job backgrounds and living conditions. Of the tests made, including liver and immunofunction studies, no differences were observed between the exposed and control groups. Although the population study is too small to permit any general conclusions as to the potential adverse health effects from long-term exposure to phenoxy acids, the investigation shows that follow-up is possible on the health status of persons exposed for several years to 2,3,7,8-tetra-CDD-containing phenoxy acids, and then to compare the exposed group results to carefully matched controls. The study is in progress.

Hexachlorophene Exposure

An excess of malformations, some severe, has been reported among children whose mothers were employed as nurses in a hospital. The mothers were exposed to hexachlorophene soap during early pregnancy, and the hypothesis of a causal relation between such an exposure and the occurrence of the malformations has been advanced (*131*); five severe and six slight malformations were observed in 65 children in the exposed group, whereas only one slight malformation was observed in the 68 children of the unexposed group. This report has been followed by another study (to be published) of a similar group of children of exposed mothers in comparison to children of unexposed mothers. Again a high frequency of malformations among the offspring of exposed mothers was reported (*132*). From these studies, however, it is not clear if exposure to other hazardous chemicals could be excluded. A retrospective-prospective study is being initiated among long-term workers of an United States' factory which used Seveso-produced 2,4,5-trichlorophenol in the manufacture of hexachlorophene. However, in the case of hexachlorophene, polychlorinated xanthenes have been also identified as contaminants at a higher level than 2,3,7,8-tetra-CDD (*133*).

Phenoxyacids and Chlorophenols Exposure in Forestry and in Wood Industry Workers

At the Regional Hospital in Umea (Northern Sweden), Hardell (*134*) observed that several patients suffering from mesenchymal tumors reported occupational or other exposures to phenoxyacids. More specifically, 87 mesenchymal tumors were diagnosed during the years 1970 through 1976. Of these cases, 55 were men (more than expected) and 43 of these 55 men were known by profession: 19 were either forestry workers, farmers and forestry workers, or workers in sawmills and papermills where exposure to chlorophenols is common. Based on the official statistics of Sweden, one can calculate approximatively the expected fraction of tumors within these trades, resulting in an expectancy of approximately 11 cases versus the 19 observed.

The Mount Sinai School of Medicine in New York is currently planning a field survey on long-term health effects on wood-preservative workers in Arkansas and Oklahoma in the United States of America.

Exposure to 2,3,7,8-Tetra-CDD during Production of 2,4,5-T or Polychlorinated Phenols and Following Industrial Accidents

The following cases, as well as others not considered at the meeting, have been described in IARC Monographs Volume 15 (*1*), and only some of the previously available information will be given here. The ongoing epidemiological studies on these cohorts are summarized in Table 7.

As each individual cohort has a relatively small number of person-years, a study has been proposed in which all the cohorts would be pooled and the mortality compared to that expected from the national statistics.

The first reported cases of industrial poisoning due to the formation of 2,3,7,8-tetra-CDD in uncontrolled exothermic reactions occurring during the manufacture of TCP were seen in 1949 at a 2,4,5-T-producing factory in Nitro, West Virginia, USA; 228 persons were affected. These individuals were studied by Suskind and Ashe (*135*) and followed up for a period of four years. Symptoms included chloracne, nausea, vomiting, headaches, severe muscular aches and pain, fatigue, emotional instability, and intolerance to cold. Laboratory findings showed raised total lipids and raised prothrombin time. Among those affected were not only workmen, but also laboratory personnel, medical personnel, and even the Safety Director who visited the areas of exposure. Several wives who had never visited the plant also developed acne, usually at the same time as their employee husbands.

It is important to note that information on the toxicity as well as on the stability (long persistence and slow degradability) of 2,3,7,8-tetra-CDD was not available at the time the first reports of acute toxicity were observed, and no measures were taken to decontaminate the factories and control the residue levels of 2,3,7,8-tetra-CDD. In fact, the presence of 2,3,7,8-tetra-CDD or other PCDDs and PCDFs was not even suspected. Workers, therefore, were probably exposed for a considerable time to 2,3,7,8-tetra-CDD following the first observations of acute toxic effects.

In November 1953, an accident occurred at a TCP factory in Ludwigshafen, Federal Republic of Germany; 75 workers were exposed to the reaction products during the accident and the subsequent cleaning work; most were affected by chloracne, 42 severely: 21 of the 42 suffered consequent damage to internal organs or disturbances of the nervous system. The most relevant features were polyneuritis, sensorial impairments, and liver damage. The son of one of the workers developed chloracne following contact with his father's work clothes. An additional case of poisoning occurred 5 years later in a worker who was involved in repair work (welding one of the auto-claves) at the contaminated site; subsequently this worker died with necrotic pancreatitis.

All the 75 workers could be traced in a cohort study 25 years later; mortality was compared both with the regional and national mortality and with a control group of workers from the same factory. In the exposed group, 17 deaths were observed (11 to 25 expected depending on the control population); six from cancer (four or less expected), five from cardiovascular diseases (as expected), two from suicides (0.2-0.6 expected), one from liver cirrhosis, one from a urogenital tract disease, and two from traffic accidents.

Of the six cancer deaths, three were from stomach cancer and occurred in the age group of 60-69, which is significantly more than expected. This effect was more pronounced when a minimum observation period of 10 years was considered, and more so for stomach cancer after a 20-year observation period. (Use of a long observation period permits a better estimation of the relation between exposure and disease when long "latency" periods are involved, as for cancers).

A similar accident occurred at the 2,4,5-T-producing factory in The Netherlands in 1963; 106 men are likely to have been exposed to 2,3,7,8-tetra-CDD (and possibly PCDFs) as they were in the building where the accident occurred during the subsequent period, March-July 1963. These workers may be divided into three groups: (A) factory employees (exposed during all phases of the cleaning and reconstruction activities), 44 men; (B) clean-up crew (nonfactory personnel, exposed during the months May-July), 18 men; and (C) plumbing, painting, and insulating personnel (nonfactory workers, exposed during the months May-July), 44 men.

During the explosion in Amsterdam, two operators were present in the building. With the exception of these men, who were probably exposed primarily by the inhalation route, all other workers used safety masks and were probably exposed by dermal contact. Chloracne was the most common and prominent lesion observed following 2,3,7,8-tetra-CDD exposure (26/44 examined in group A, 10/16 in group B, and 8/16 in the group C). Liver function tests (thymol turbidity and serum glutamic pyruvic transaminase and serum glutamic oxaloacetic transaminase) did not indicate liver damage. A few men complained of fatigue. The latent period between exposure and the appearance of chloracne, characterized by comedones, pustules, and cysts on the face and sometimes on other parts of the body, was approximately 4-6 weeks. Sometimes, erythematous and edematous skin lesions, possibly due to phenolic compounds, were noted one day after exposure. No skin lesions (chloracne) were reported in family members of the employees. Of group A, 3/44 workers died, one of pancreas carcinoma in 1964, one of myocardial infarction at the age of 69, and one of a traffic accident. Of the 17/18 traced individuals in the B group, four have died (three from sudden death, probably of myocardial infarction, at ages, 41, 53, and 65, and one of ill-defined pulmonary or cardiac cause at age 44). Only 32/44 of the C group were traced, and one of them died of sudden death, probably myocardial infarction, at age 50.

Jirásek et al. (*104*, *105*, *109*) studied 55 subjects from a cohort of 78 workers from a factory in Czechoslovakia producing 2,4,5-T and pentachlorophenol, who were affected by chloracne. The workers were probably exposed during the years 1965-1968 to unknown levels of 2,3,7,8-tetra-CDD. A significant symptom was the disturbance in porphyrins metabolism with signs of porphyria cutanea tarda. Many workers also suffered severe hepatic and neurological damage and had raised blood levels of cholesterol and total lipids. In the first years of follow-up, five deaths were observed, two from bronchogenic carcinoma at age 47 and 59 (only 0.12 lung cancer deaths were expected from national mortality statistics), one from atypical atherosclerosis at age 57, and one from the acute occupational intoxication. One further death, probably from liver cirrhosis, has recently been observed and 50 workers are still being followed. Many workers continue to show lipemia and hypercholesterolemia and have developed prediabetic changes and hypertension. The workers lost to follow-up are mostly foreigners who returned to their country of origin.

In July 1976, an accident in a TCP-producing factory in Meda, Italy resulted in the contamination of the towns of Seveso, Meda, Cesano Maderno, Desio, Nova Milanese, and possibly others of a large, densely populated area called the Brianza di Seveso with a total population of about 220,000 inhabitants. A retrospective health survey conducted on the workers of the ICMESA factory revealed that two workers among those directly involved in the production of 2,4,5-trichlorophenol suffered from skin lesions previous to July 1976. These facts may be indicative of a previous 2,3,7,8-tetra-CDD exposure.

A large and intensive follow-up program is in progress in the Seveso area; data were presented about the first dermatological, neurological, clinical, and laboratory findings, as well as about fetal damage. Dermatological examinations, including the screening of 32,000 children, resulted in the finding of hundreds of children with skin lesions, 135 of which were eventually identified as chloracne; 59 cases were from the more contaminated area (Zone A). However, precise description of diagnostic criteria and accurate anamnestic information, including, among others, data on places of residence, were not included. This information is needed before evaluating the meaning of the chloracne incidence data by territorial areas and efforts should be made obtain it. Chloracne cases were not systematically searched for in adults. Neurological examinations showed both signs of subclinical neurological damage and cases of clinically detectable polyneuropathy in adults (34/338 persons from zone A and 8/185 from the surrounding areas).

In about 30% of 1654 adults from both A zone and nearby zones, hepatomegaly was reported to be present on clinical investigation. No information, however, is given on the criteria by which the hepatomegaly was evaluated. The same percentage of abnormalities was observed in one or more liver tests (mainly glutamyl transferase and serum glutamic pyruvic transaminase and serum glutamic oxaloacetic transaminase). Extensive monitoring of various hematological parameters from 25,000 persons is now in progress and the data are expected to become available during 1978. Results of this investigation should allow a better understanding as to whether or not these alterations are caused by exposure to 2,3,7,8-tetra-CDD and PCDDs.

So far, immunological investigations, cytogenetic research, and embryomorphology analyses on cases of therapeutical abortions have not given abnormal results. However, the study of the frequency of congenital malformations and spontaneous abortions seems to be very difficult to accomplish. The importance of a systematic study on this subject has been stressed by the Working Group, because the questions on the teratogenicity of 2,3,7,8-tetra-CDD in humans cannot be solved by any other study.

A preliminary mortality study has been possible in 2 of the 11 towns of the Seveso area. The 1975-1977 mortality rates from liver cirrhosis and leukemia in these two limited zones of the Seveso area were compared to that of a nearby area (province of Varese). Within an overall mortality which did not appear different, an increase of deaths from liver cirrhosis and from leukemia was noted. The validity and significance of these observations should be carefully evaluated.

Recommendations

A chemical analytical program should be encouraged to identify and standardize analytical

Table 2. Epidemiological studies on 2,3,7,8-tetrachlorodibenzo-p-dioxin; human exposure episodes caused by accidental or occupational exposure occurring in factories producing 2,4,5-trichlorophenol and/or 2,4,5-trichlorophenoxyacetic acid.

Town and country	Year of exposure	Number of exposed persons	Clinical follow-up and cohort studies	Observations to date	Proposed actions	Comments and recommendations of the NIEHS/IARC Working Group
Accidental exposures						
Nitro, West Virginia, (U.S.A.)	1949	228	Only 36 individuals were followed, for a period of 4 years	No significant long-term observations are yet available (some followed-up individuals had symptoms of severe intercostal neuritis	Mortality cohort study being planned	Efforts should be made to trace the individuals who were exposed, possibly through the employment compensation files.
Ludwigshafen/Rhein, Federal Republic of Germany	1953	75	A mortality cohort study has conducted 24 yr after the accident; all exposed workers were traced for a total of 1525 person-years of observation	6 Cancer deaths were observed: 3 of stomach cancer in the age group 60–69 (significantly higher than expected), 2 of oat-cell carcinoma of the lung, and 1 of colonic adenocarcinoma	Follow-up to be continued	The results of the continuing follow-up study should periodically be made available.
Amsterdam, The Netherlands	1963	106	Preliminary observations on mortality are available; 93 exposed workers have been traced up to 1977	Of 8 deaths, 5 or 6 were from cardiovascular diseases, 5 probably from myocardial infarctions; the myocardial infarction death rate seems higher in the most heavily exposed workers affected by chloracne. One worker, who did not develop chloracne, died from pancreatic carcinoma 14 months after the accident; however, he complained of abdominal pain prior to the accident	Full report planned for 1978	Efforts should be made to trace all the workers who were exposed.
Bolsover, Derbyshire, United Kingdom	1968	90	Of the workers belonging to the original cohort, 50% are still employed and routinely undergo clinical and laboratory investigations	One worker is known to have died from coronary thrombosis	Epidemiological cohort study to be planned when the results of the present clinical follow-up investigation become available	Efforts should be made to trace all the workers who were exposed.

Meda, Brianza di Seveso, Italy	1976	?[a]	A clinical follow-up of the inhabitants from the more contaminated areas is being carried out. At the time of the accident, living in Zone A was a population of 730 people, with 200 of those being between 0-14 years of age. Of these 730 persons, 623 (125 between 0-14 years of age) were followed-up. Zone B contained a population of 4732 inhabitants with 1293 between the ages 0-14 years. An accurate count of those followed in Zone B is not available, but 943 youngsters between 0-14 years of age are included in the follow-up	Initial clinical observations showed a high incidence of 2,3,7,8-tetra-CDD-dependent skin lesions among children living in the more contaminated area. Also, a number of persons residing in the zone surrounding the more contaminated areas had skin lesions, but with poorly defined, clinical features; these lesions may be the result of 2,3,7,8-tetra-CDD exposure. Moreover, both clinical and laboratory findings suggest an increased incidence of hepatic sufferance among adults living in the highly contaminated areas. A preliminary mortality study suggests that although the overall rates from 2 Seveso towns are not different from a nearby area, an increase of deaths from liver cirrhosis and leukemia was noted	Among other ongoing investigations, a long-term morbidity study has been initiated	Possible teratogenic and other adverse reproductive and developmental effects from 2,3,7,8-tetra-CDD exposure should be investigated. A mortality register and population files, suitable for adequate descriptions of morbidity data, should be established. Further, the cancer registry that already covers a nearby province, should be extended to include the Seveso area.
Occupational exposure						
Czechoslovakia	1965-1969	80	A follow-up has been made regularly on 55 workers of the original cohort. The workers lost to follow-up are mainly foreigners who left the country	Five patients died during the first 5 year-follow-up period: 2 died from bronchogenic carcinoma and 1 from a rapidly developing atypical atherosclerosis. One further death, probably from liver cirrhosis, has recently occurred. Many of the workers are now suffering from hypertension and show signs of hyperlipidemia and hypercholesterolemia as well as prediabetic symptoms and signs	Follow-up to be continued. A 10-year report will be prepared during 1978	The results of the continuing follow-up study should periodically be made available.

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[a] No reliable information was available to the Working Group about the actual number of exposed persons; the entire area has a population of approximately 200,000 people.

methods used, to catalogue products contaminated with PCDDs and PCDFs, and to establish a register of known PCDD and PCDF standard samples.

The Working Group unanimously recommended that a system be developed for an international exchange of information and research coordination on the health effects of chlorinated dibenzo-*p*-dioxins/chlorinated dibenzofurans. Further, and in particular, the following three areas were indicated as deserving special attention:

1. Development of sensitive and reliable measurements of body burden; improved, more sensitive, and less expensive analytical technology should be generated. Standardized sampling (air, water, soil, tissues and body fluids, environmental), preparation, clean-up, and analysis should be promoted;
2. Development of common protocols for clinical examinations (including reproductive experience) and, in particular, design of a protocol for basic core information;
3. Development of an international registry of exposed persons to serve as a basis for long-term follow-up: this was considered especially important because the relatively small size of populations involved in individual exposure episodes are an obstacle to risk assessment and thus pooling of data is almost a necessity.

The Working Group recommended that IARC explore the technical and financial feasibility, possibly in cooperation with other national and/or international organizations, of setting-up such a permanent mechanism which could include as a minimum starting program: (a) a periodical, perhaps biennial, meeting to up-date the progress of ongoing studies, and to identify needed developments; (b) an exchange of relevant information accruing during the interval between meetings; and (c) a register of chloracne cases, containing simple, essential information in a standardized form. This PCDDs/PCDFs exposure-related sign may help to better define an exposed cohort population suitable for long-term follow-up.

This report represents the views and opinions of the U.S. NIEHS/IARC (National Institute of Environmental Health Sciences/International Agency for Research on Cancer *ad hoc* Working Group which met January 10-11, 1978 in Lyon France to review the history of human exposure to these chemicals, to collate current information, and to plan and forecast needed new directions. The meeting was funded jointly by NIEHS and IARC.

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ENVIRONMENTAL CONCENTRATIONS OF LEAD AND CADMIUM IN THE AREAS OF SECONDARY LEAD SMELTERS AND A BATTERY MANUFACTURING PLANT IN DALLAS, TEXAS

EPA CONTRACT NUMBER Ot - 0918 - NTEX

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July 1, 1981
ERRATA SHEET—

s Larry L. Thom
ficer

OM IT MAY CONCERN

The following additions and corrections are provided as an addendum to the report titled "Environmental Concentrations of Lead and Cadmium In The Areas of Secondary Lead Smelters and A Battery Manufacturing Plant in Dallas, Texas" EPA Contract Number Ot - 0918 - NTex.

1. In every place throughout the report where the name Dixie Metal Company (DMC) is used, it should be corrected to read Dixie Metal Company/N.L. Industries (DMC/NL). The N.L. plant is located across the street from the DMC plant. N.L. was closed in 1979.

   On Page 2

   The address for N.L. Industries should be added: 1000 Sargent Road.

   On Page 17

   Add a * to 14-HS 1 and 2 (25,503.0* and 24,851.0*)

   At bottom of page add:
   * These values are abnormally high and could be due to contamination caused by the owner of the house. The contractor is resampling this area.

   On Page 30

   The average value around DMC should be changed to read as follows:
   1253.66 (680-720)*
   * This average results from deleting or replacing the 24,000 and 25,000 values.

   On Page 34

   The average around the Reference area should read 54 ppm.
   Eight sample areas in RSR area contained greater than 1000 ppm lead.
   RSR-10-HS was missed.

   On Page 38

   DMC-18 2608 Gould

   On Page 39

   RSR-9 Carver Elementary School, Corner of Holystone and Bickers

ABSTRACT

The object of this research project was to characterize environmental concentrations of lead and cadmium in the area of secondary lead smelters and lead-acid battery manufacturing plants. Particular attention was devoted to collecting samples from areas where children would be expected to be exposed to these environmental pollutants.

Selected households downwind from two secondary lead smelters and one battery manufacturing plant were sampled for lead and cadmium concentrations in hot and cold house tap water, dust on refrigerator tops and window sills, in front yard and back yard soil as well as garden soil if a garden existed on the premisses. Additionally, a plastic dustfall tray was left in an undisturbed area, usually the top of the refrigerator, for 28-days.

A similar sampling protocol was conducted in a reference area which was similar to the study areas except it did not contain any secondary lead smelters or battery manufacturing plants. It also was chosen so that winds would not carry emissions from these type operations into the reference area.

The average concentration of lead in household tap water was slightly higher in the reference area than in the study areas, 1.59 ppb lead vs. 1.58, 0.64, and 1.35. Thus there appeared to be no impact on the study area household tap water concentrations of lead due to the plant operations.

However, 28-day dustfall, dust wipe and soil lead concentrations were all significantly higher in the study areas when compared to the reference area.

Cadmium levels in house tap water samples were all of the same order of magnitude and did not indicate any impact from smelter or battery plant operations. The concentrations were all quite low, ranging from below the detection limit of about 0.01 ppb to 0.30 ppb.

The 28-day dustfall trays indicated levels of about the same magnitude of cadmium in all areas. However, refrigerator dust wipes indicated levels averaging about 35% higher in the study areas compared to the reference area. This relationship was not continued with the window sill wipes. The study areas all had lower cadmium concentrations than the reference area, or average. There was no reason to suspect any difference in the two areas due to paint as the economic level of the areas approved to be about the same.

Concentrations of cadmium in soil samples ranged from 2 to 8 times greater in the study areas compared to the reference area. Thus soil cadmium levels appeared to be affected by operations of secondary lead smelters or lead-acid battery manufacturing operations.

TABLE OF CONTENTS

Page

ABSTRACT i

INTRODUCTION 1

SAMPLE SITES 2

SAMPLING PROTOCOL 11

ANALYSIS PROTOCOL 13

LEAD AND CADMIUM CONCENTRATIONS 15

CONCLUSIONS 32

APPENDICES 36

APPENDIX A. SITE LOCATIONS 37

APPENDIX B. SITE PICTURES 41

APPENDIX C. SAMPLE COLLECTION SCHEDULE 80

APPENDIX D. WEATHER DATA 82

LIST OF TABLES

TABLE PAGE

1 Pb Soil Samples RSR Area 16

2 Pb Soil Samples DMC Area 17

3 Pb Soil Samples GBC Area 18

4 Pb Soil Samples REF Area 19

5 Cd Soil Samples RSR Area 20

6 Cd Soil Samples DMC Area 21

7 Cd Soil Samples GBC Area 22

8 Cd Soil Samples REF Area 23

9 Pb Dust Tray Samples 24

10 Cd Dust Tray Samples 25

11 Pb Dust Wipe Samples 26

12 Cd Dust Wipe Samples 27

13 Pb House Water Samples 28

14 Cd House Water Samples 29

15 Cadmium Data Summary 30

16 Lead Data Summary 31

C-1 Data Samples Collected 81

LIST OF FIGURES

FIGURE PAGE

1 RSR Corporation (RSR) Soil Sample Sites 2

2 General Battery Corporation (GBC) Soil Sample Sites . . 3

3 Dixie Metal Company (DMC) Soil Sample Sites 4

4 Reference Area (REF) Soil Sample Sites 5

5 RSR Corporation (RSR) Soil Sample Sites 6

6 General Battery Corporation (GBC) House Sample Sites . . 7

7 Dixie Metal Company (DMC) House Sample Sites 8

8 Reference Area (REF) House Sample Sites 9

9 Average Wind Direction Dallas Love Field 10

LIST OF PICTURES

PICTURE PAGE
1. DMC Site 1 42
2. DMC Site 2 42
3. DMC Site 3 43
4. DMC Site 4 43
5. DMC Site 5 44
6. DMC Site 6 44
7. DMC Site 7 45
8. DMC Site 8 45
9. DMC Site 9 46
10. DMC Site 10 46
11. DMC Site 11 47
12. DMC Site 12 47
13. DMC Site 13 48
14. DMC Site 14 48
15. DMC Site 15 49
16. DMC Site 16 49
17. DMC Site 17 50
18. DMC Site 18 50
19. DMC Site 19 51
20. DMC Site 20 51
21. GBC Site 1 52
22. GBC Site 2 52
23. GBC Site 3 53
24. GBC Site 4 53
25. GBC Site 5 54

PICTURE PAGE
26. GBC Site 6 54
27. GBC Site 7 55
28. GBC Site 8 55
29. GBC Site 9 56
30. GBC Site 10 56
31. GBC Site 11 57
32. GBC Site 12 57
33. GBC Site 13 58
34. GBC Site 14 58
35. GBC Site 15 59
36. GBC Site 16 59
37. GBC Site 17 60
38. GBC Site 18 60
39. GBC Site 19 61
40. GBC Site 20 61
41. RSR Site 1 62
42. RSR Site 2 62
43. RSR Site 3 63
44. RSR Site 4 63
45. RSR Site 5 64
46. RSR Site 6 64
47. RSR Site 7 65
48. RSR Site 8 65
49. RSR Site 9 66
50. RSR Site 10 66
51. RSR Site 11 67
52. RSR Site 12 67

PICTURE PAGE
53. RSR Site 13 68
54. RSR Site 14 68
55. RSR Site 15 69
56. RSR Site 16 69
57. RSR Site 17 70
58. RSR Site 18 70
59. RSR Site 19 71
60. RSR Site 20 71
61. REF Site 1 71
62. REF Site 2 71
63. REF Site 3 72
64. REF Site 11 72
65. REF Site 12 73
66. REF Site 13 73
67. REF Site 14 74
68. REF Site 15 74
69. REF Site 16 75
70. REF Site 17 75
71. REF Site 18 76
72. REF Site 19 76
73. REF Site 20 77
74. RSR Corporation 77
75. General Battery Corporation 78
76. General Battery Corporation 78
77. Dixie Metals Company 79

INTRODUCTION

Lead is a ubiquitous environmental pollutant with known toxic effects to exposed persons. Considerable study has shown that children below the age of 5 years are most susceptible to adverse health effects when exposed to significantly elevated environmental concentrations of lead.

Cadmium, another heavy metal, has not undergone as extensive an environmental study as lead. However, due to the use of cadmium in many processes and coatings, there is a growing concern about environmental cadmium levels. Cadmium exhibits known toxic effects and has been linked to poisoning due to environmental exposure.

This study investigated the presence in the environment of lead and cadmium. Concentrations in the area of secondary lead smelters and battery manufacturing plants were the specific topic of this project. Additionally, emphasis was placed on sampling areas where children would most likely be exposed.

Soil samples were collected from front and back yards of private residences in areas downwind from secondary lead smelting operations and battery manufacturing plants. Samples of hot and cold house tap water were also taken, in addition to dust wipes of refrigerator tops and window sills. A dust tray was also left at these homes for 28-days, after which the trays were analyzed for lead and cadmium dustfall.

Soil samples were also collected from parks, playgrounds, elementary school playgrounds and day care center playgrounds downwind from secondary lead smelters and battery manufacturing plants. A similar sampling program was conducted at houses, parks, playgrounds, and elementary school playgrounds in a reference area with no known significant exposure to emissions from secondary lead smelter and battery manufacturing operations.

SAMPLE SITES

Samples of soil, house dust and tap water were collected in neighborhoods within a two mile radius of the following locations in Dallas, Texas:

RSR Corporation (RSR)
111 W. Mockingbird

Dixie Metal Company (DMC)
3030 McGowen

General Battery Company (GBC)
2040 Amelia Street

St. Augustine Park (REF)
St. Augustine

The first three sites above were considered to be potential lead and cadmium source sites. The last site was selected as a reference or baseline area.

Figures 1, 2, 3 and 4 depict the locations of elementary schools, day care centers and playgrounds that were sampled. Figures 5, 6, 7 and 8 show the locations of all houses from which samples were obtained. Sample site addresses and pictures are contained in Appendix A. Sample sites were randomly selected in a downwind location in accordance with a wind rose developed for Dallas Love Field, figure 9.

The reference area was selected to be in the South Eastern quadrant of Dallas for three main reasons. This area has no known sources of lead or cadmium other than vehicles. There are no major federal or interstate highways that intersect the region selected. And finally, the prevailing winds are such that the transport of air masses from known source sites to the reference area was minimized.

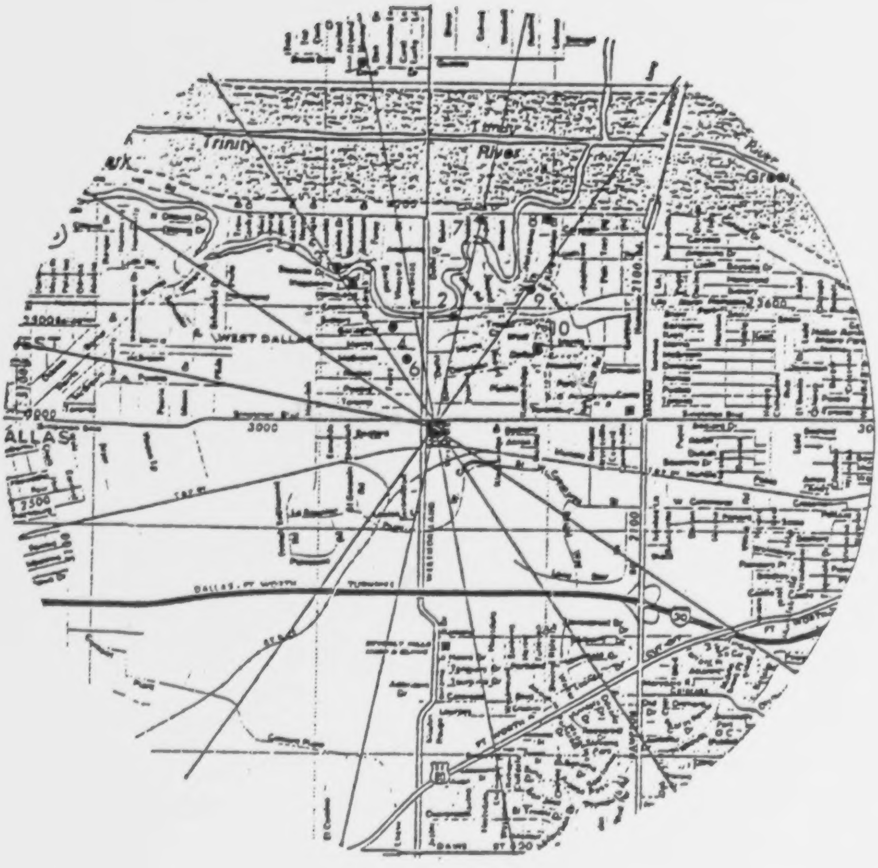

Trinity
Rivers
Trinity
River
Green
WEST DALLAS
ALLAS
3000
2500
2100
2600
30
DALLAS-FT. WORTH TURNPIKE
1
2
6
7
8
9
10

FIGURE 1

RSR CORPORATION (RSR) SOIL SAMPLES SITES

(EXCLUDING HOUSE SAMPLE SITES)

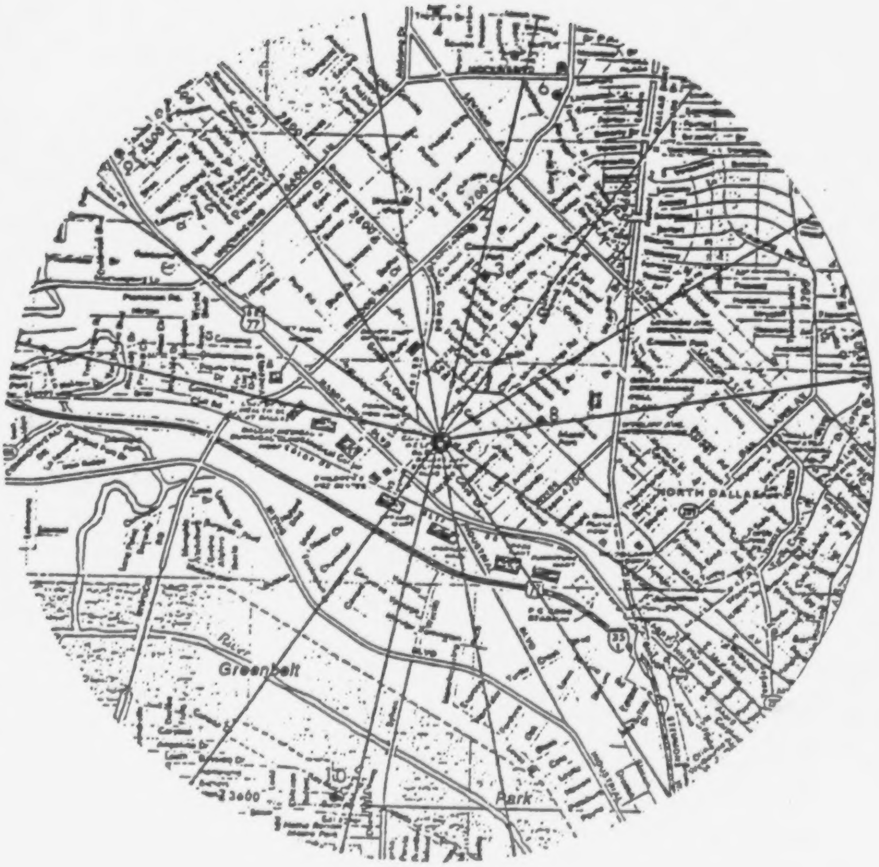

NORTH DALLAS
Greenbelt
River
Park
77
35

FIGURE 2

GENERAL BATTERY CORPORATION (GBC) SOIL SAMPLE SITES

(EXLCUDING HOUSE SAMPLE SITES)

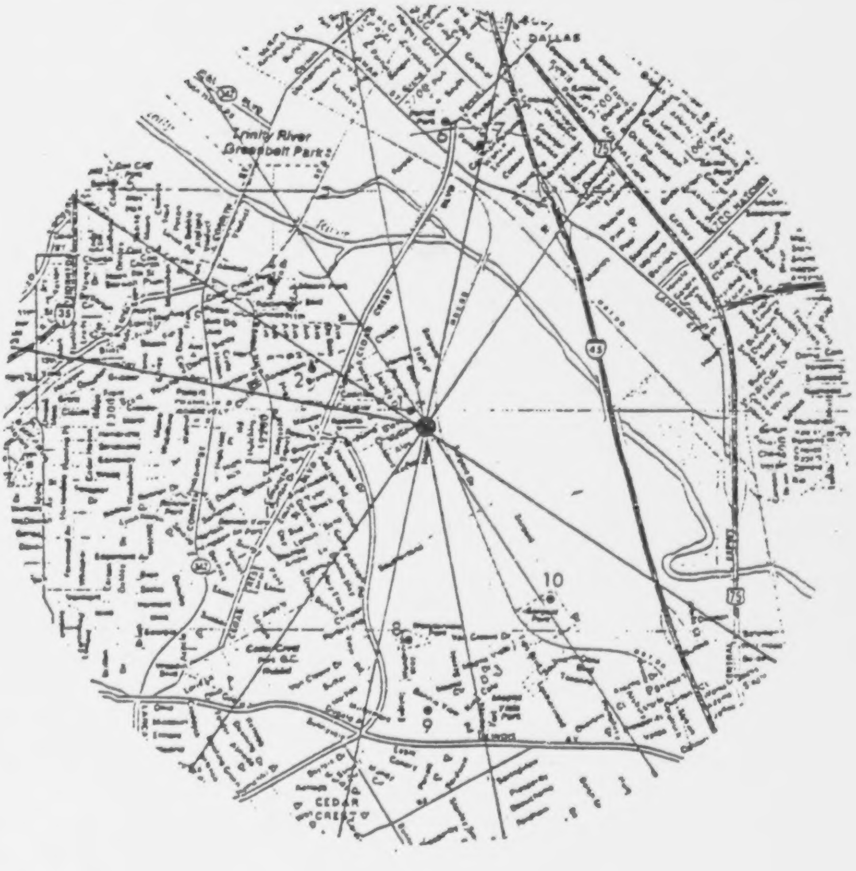

DALLAS
Trinity River
Greenbelt Park
2
8
9
10
CEDAR CREST
35
45
75

FIGURE 3

DIXIE METAL COMPANY (DMC) SOIL SAMPLE SITES (EXCLUDING HOUSE SAMPLE SITES)

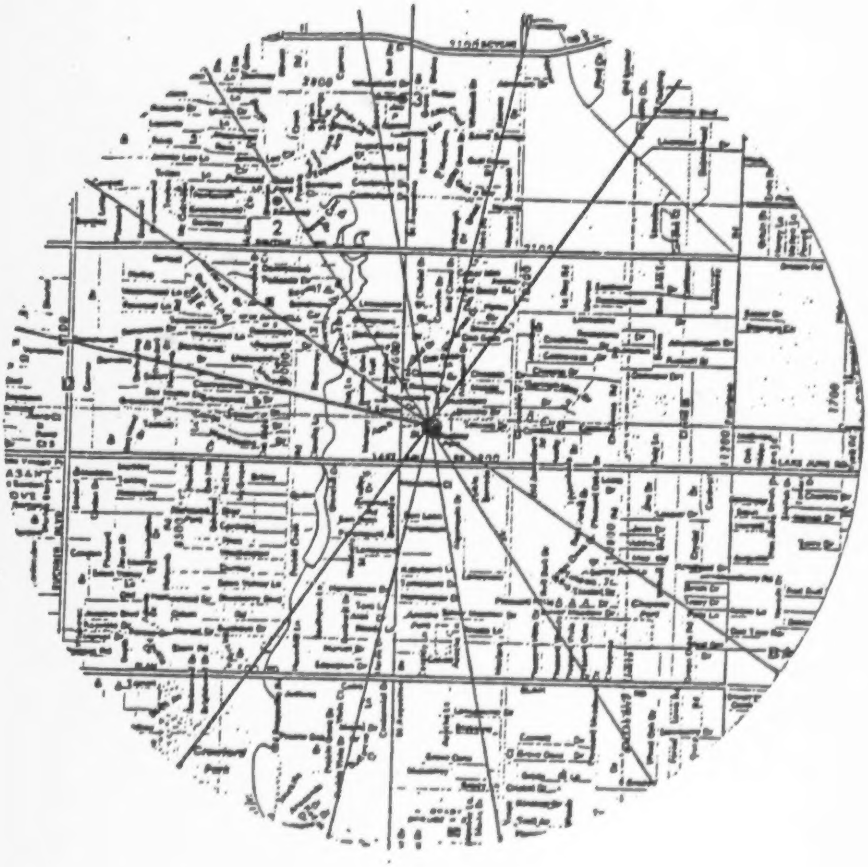

2
3
3
LAKE JUNE RD

FIGURE 4

REFERENCE AREA (REF) SOIL SAMPLES SITES

(EXCLUDING HOUSE SAMPE SITES)

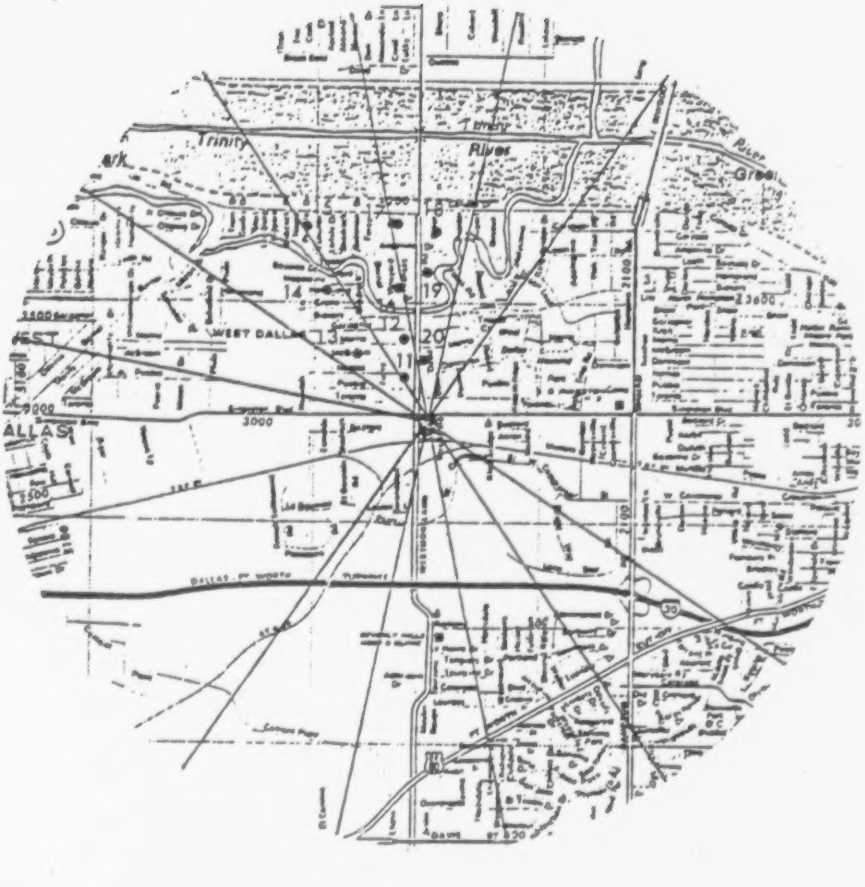

Trinity
River
Trinity
Green
West Dallas
WEST
ALLAS
14
19
12
13
20
11
3000
2100
3600
2500
3000
DALLAS - FT WORTH
30

FIGURE 5

RSR CORPORATION AREA (RSR) HOUSE SAMPLE SITES

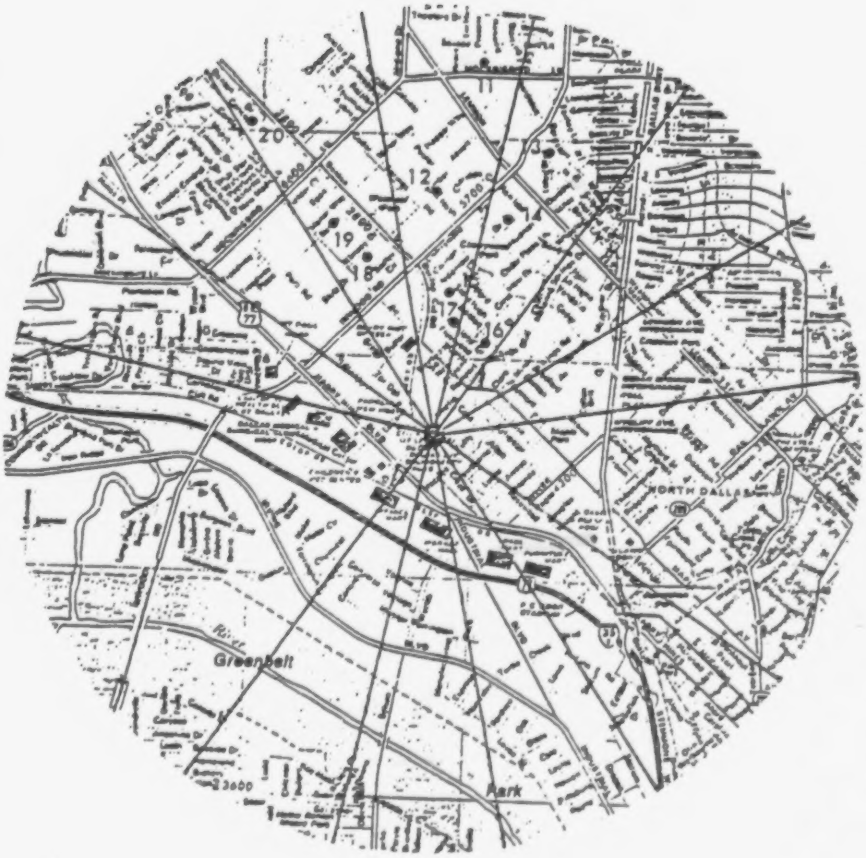

20
12
3
14
19
18
17
16
NORTH DALLAS
River
Greenbelt
Park

FIGURE 6

GENERAL BATTERY CORPORATION (GBC)

HOUSE SAMPLE SITES

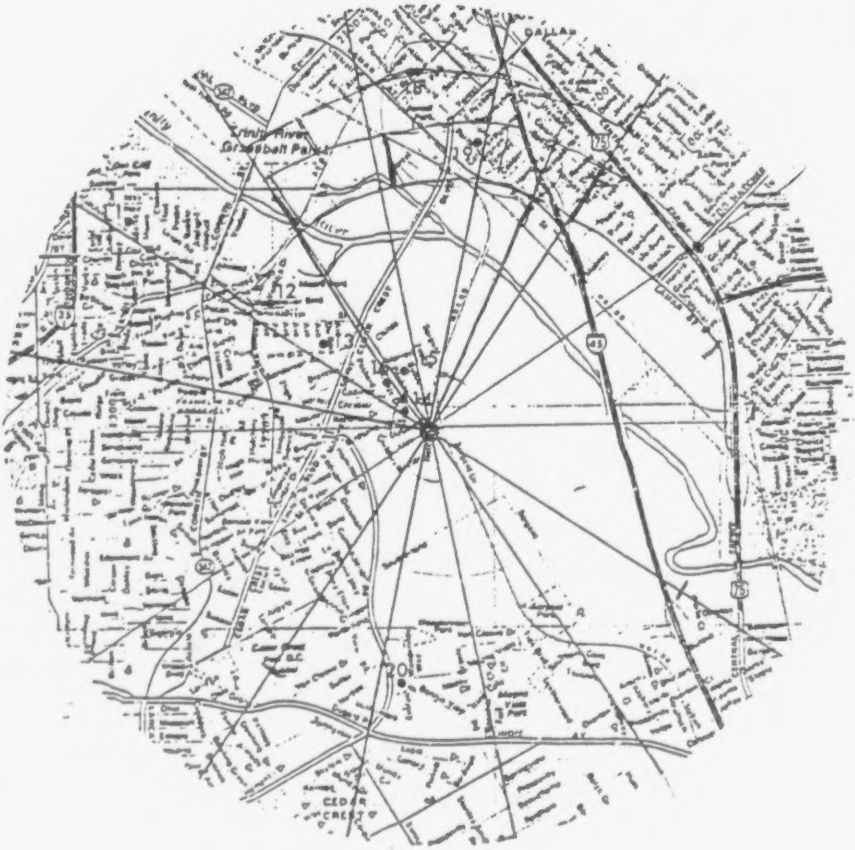

DALLAS
Trinity River
Greenbelt Park
75
45
12
13
28
20
CIDAR
CREST

FIGURE 7

DIXIE METAL COMPANY (DMC) HOUSE SAMPLE SITES

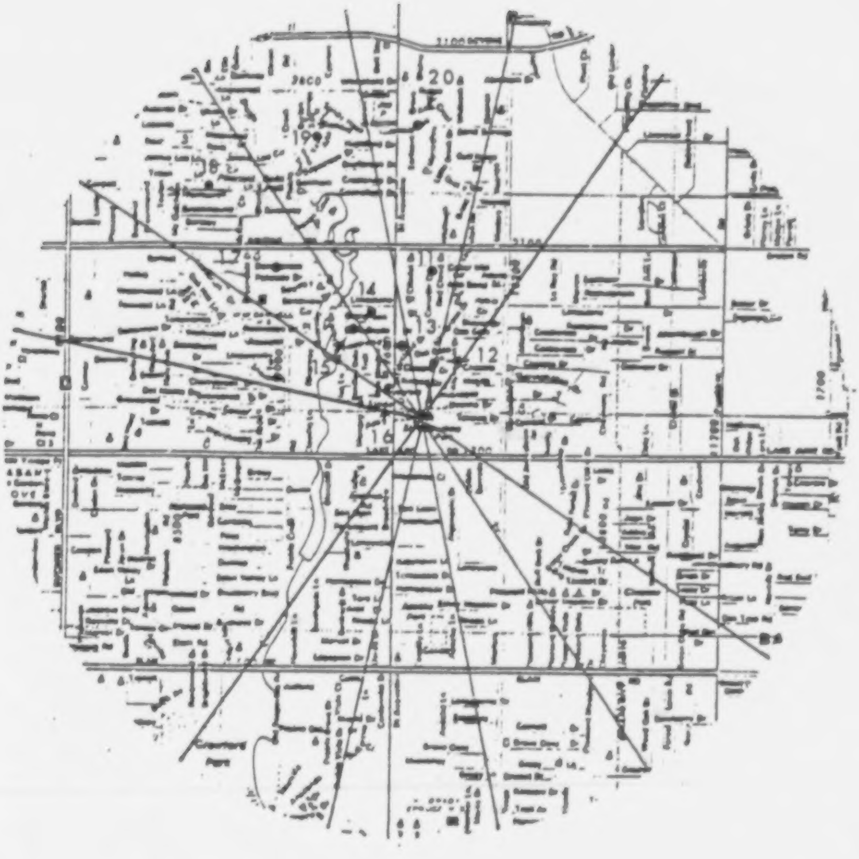
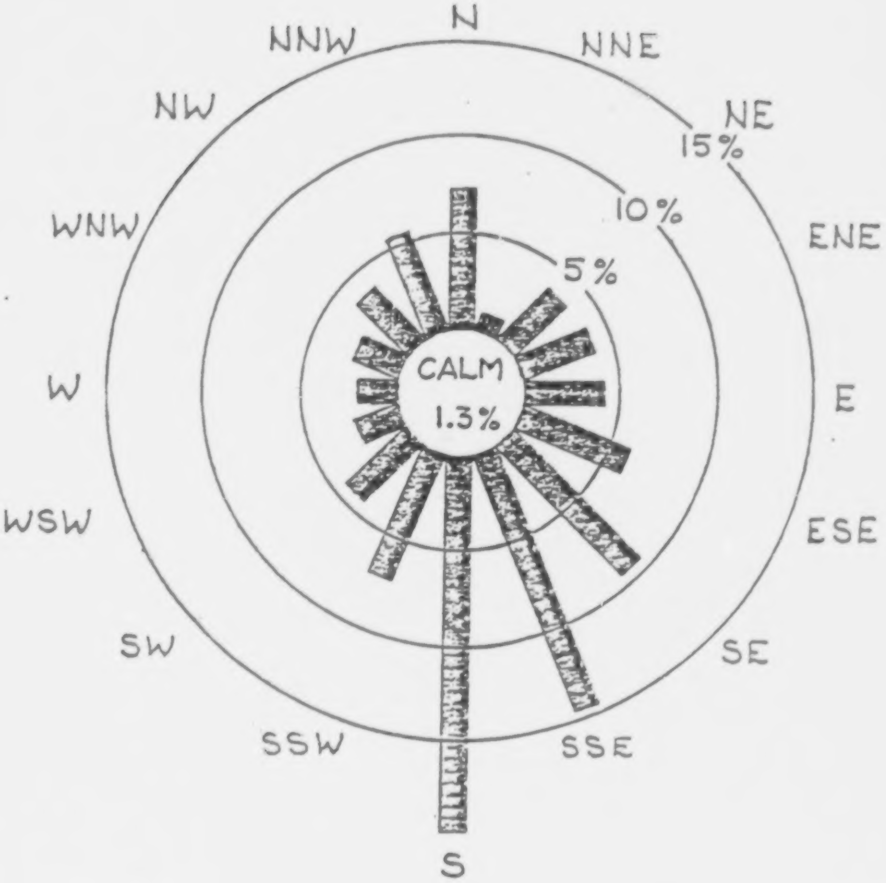

20
19
18
14
13
12
15
16

FIGURE 8

REFERENCE AREA (REF) HOUSE SAMPLES SITES



AVERAGE WIND DIRECTION
DALLAS LOVE FIELD 1961~1973

ARGENTO
10-21-198

FIGURE 9

SAMPLING PROTOCOL

Soil samples were collected from all sites. A minimum of four soil samples was collected at each site. At house sites, samples were collected, when possible, two from the front yard, two from the back yard and two from any garden that existed. All soil samples were obtained with a stainless steel core device that produces a ground surface core two inches in diameter and three inches tall. All soil samples were placed in polyethylene containers and stored at room temperature to await Pb and Cd analysis.

Water samples were collected in 500 ml acid-washed polyethylene bottles, one each from the hot water and cold water tap in the kitchen of each house. Water was allowed to run from the kitchen taps for one minute before the samples were collected. Each sample bottle was then rinsed with either hot or cold tap water three times before a sample of hot or cold water was collected, respectively. The samples were then acidified (1%) within 90 minutes using concentrated HNO_3 (reagent grade). The samples were then stored at room temperature until analyzed.

Housedust samples were collected utulizing two different techniques. The first technique used an acid-washed linear polyethylene tray as the sampling vessel. The tray was left in the house for 28 days on the top of the refrigerator in the kitchen. Upon collection, the tray was sealed with an acid washed polyethylene lid. The contents of the tray were then not disturbed until extraction procedures began.

The second technique for dust utilized wipes (eg - a commercial towelette, Diaparene). This above mentioned brand was selected because they were all dispensed from the same solution from a polyethylene container and was, therefore, more likely to be consistant in chemical makeup. Two wipes were taken at each house from a one hundred square centimeter area. A polyethylene template was used to insure the area size. One wipe was from a kitchen window sill (5 cm x 20 cm) and the other wipe was from the top of the refrigerator (10 cm x 10cm). The towelettes were placed in self-sealing polyethylene bags after sampling to await analysis. All dust wipe sampling was performed while wearing clean disposable polyethylene gloves that were discarded after each sample.

ANALYSIS PROTOCOL

Soil samples were dried overnight at 65 degrees centigrade in acid-washed stainless steel pans in an electric oven. The dried soils were then ground to powder using a glass mortar and pestal and sieved through a 250 micron stainless steel screen. A 5 gram sample of the powdered soil was then weighted into an acid-washed 125 ml Erlenmeyer flash and 50 ml of leach solution was added. The leach solution was a 7:3 ratio by volume of 35% acetic acid by volume and 25% aquious solution of hydroxylamine hydrochlorine by volume. The flask was then sealed using a polyethylene stopper and the mixture shaken for 12 to 14 hours on a mechanical shaker.

The mixture was then filtered through a glass fiber filter (9cm) which was prerinsed with leach solution. The filtrate was then collected in 50 ml acid-washed polyethylene bottles and lead cadmium concentrations were determined using flame atomic absorption spectrophotometry (AAS). Flame AAS was used because of high concentration.

Water samples were analyzed directly for lead and cadmium using flameless AAS.

Dust trays were rinsed with distilled water into a 250 ml Vycor beaker. Twenty-five ml of concentrated HNO_3 (Baker ultra pure for trace metals) was then used to rinse the dust tray and added to the Vycor beaker. The beaker was placed on a hot plate (150 degrees centigrade). A raised watch glass was placed on the beaker during digestion. The sample was heated without boiling until only 2 to 3 ml remained. The sample was then cooled and quantitively transferred to a graduated centrifuge tube (12 ml) and adjusted to 5 ml with deionized water. After centrifuging for 30 minutes at 3000 RPM, the supernatant was decanted into a small vial without disturbing the silica precipitate. The solution was then analyzed for lead or cadmium using a flame AAS.

The dust wipe samples were analyzed after digesting the towelettes. The towelettes were removed from their bags and placed in a 2.5 cm by 8.5 cm extraction thimble. Eighty (80) milliliters of the digestion acid (15 ml of HCL (Barker ultra pure for trace metals) and 65 ml of HNO_3 (Barker ultra pure for trace metals)) were then added to the 125 ml boiling flask of a Bailey-Walker extraction apparatus. The digestion acid was then heated at reflux temperature for three hours. The solution was then cooled and 10 ml of 0.1 HNO_3 were added through the condenser. After the acid drained through the thimble, the thimble was removed from the boiling flask with acid-washed stainless steel tongs. The flask was placed on a hot plate (300 degrees Centigrade) and the solution evaporated. After evaporating to 2 to 3 milliliters the flask was removed, cooled and quantitively transferred to a graduated centrifuge tube (15 ml). The volume was made to 5 ml with distilled water and the mixture centrifuged for 30 minutes at 2000 RPM.

A portion of the supernatant was decanted into a polyethylene vial (5 ml) without disturbing the precipitate. This solution was then analyzed for lead and cadmium using flame AAS.

All glassware, polyethylene and utensils used in the analysis procedures were acid-washed in concentrated HNO_3 before their use.

Quality assurance procedures included: the use of reagent blanks; determination of recovery factor for each type of extraction; replication of 10 percent of all AAS readings; maintenance of AAS linear analysis region by using calibration curve and monitoring standard solutions every tenth sample at a minimum; and the diluting of samples if outside linear concentration region.

LEAD AND CADMIUM CONCENTRATIONS

The lead and cadmium concentrations found in the soil are tabulated in Tables 1, 2, 3, 4, 5, 6, 7, and 8. The sample number in these tables are coded to show the site function as follows:

PS	Playground Soil
SS	Elementary School Soil
KS	Kindergarten Soil
DS	Day Care Center Soil
HS	House Soil
GS	House Garden Soil

The lead and cadmium concentrations for the 28 day dust tray/dust fall samples are shown in Tables 9 and 10.

Lead and cadmium concentrations for the house dust wipe samples from window sills and refrigerators are tabulated in Tables 11 and 12.

Lead and cadmium concentrations observed in both hot and cold water are tabulated in Tables 13 and 14.

Table 15 is a summary of cadmium concentrations by sample type showing the average sample concentration, $\overline{x}$, and standard deviation, S.D., for each source area. Table 16 is a summary of lead concentrations by sample type showing the average sample concentration, $\overline{x}$, and standard deviation, S.D., for each source area.

TABLE 1

Pb SOIL SAMPLES
RSR AREA
(µg of Pb/g of Soil)

SAMPLE NUMBER	1	2	3	4
1 - PS	92.8	45.8	70.3	81.6
2 - PS	256.6	215.2	311.1	279.1
3 - PS	113.5	173.8	331.8	160.6
4 - SS	690.1	669.4	256.6	194.5
5 - PS	1435.3	119.2	292.3	196.3
6 - DS	1683.7	335.6	292.3	1145.5
7 - PS	273.5	111.7	205.8	196.3
8 - DS	72.1	62.7	38.3	42.0
9 - SS	98.5	85.3	149.3	85.3
10 - SS	669.4	141.8	1228.3	1559.5
11 - HS	1000.6	1228.3	1249.0	1352.5
12 - HS	1870.0	1828.6	1104.1	1104.1
13 - HS	1228.3	1000.6	835.0	1000.6
14 - HS	136.1	158.7	235.9	288.6
15 - HS	55.2	109.8	194.5	130.5
16 - HS	237.0	337.5	271.6	382.6
17 - HS	230.2	130.5	87.2	151.2
18 - HS	124.8	30.7	177.5	254.7
19 - HS	350.6	181.3	1331.8	158.7
20 - HS	959.2	1269.7	1021.3	1248.9

TABLE 2

Pb SOIL SAMPLES
DMC AREA
(μg of Pb/g of Soil)

SAMPLE NUMBER	1	2	3	4
1 - PS	1145.5	1663.0	1435.3	1207.6
2 - PS	128.6	106.0	119.2	139.9
3 - PS	361.9	53.3	202.0	241.5
4 - PS	107.9	115.4	98.5	107.9
5 - PS	59.0	32.6	47.7	70.3
6 - PS	153.1	194.5	196.3	186.9
7 - SS	89.1	77.8	53.3	55.2
8 - PS	17.6	15.7	68.4	74.0
9 - SS	43.9	32.6	53.3	51.4
10 - PS	89.1	79.7	70.3	64.6
11 - HS	211.4	183.2	731.5	1249.0
12 - HS	107.9	121.1	273.5	329.9
12 - GS	320.5	177.5		
13 - HS	158.7	256.6	290.4	264.1
14 - HS	25,503.0	24,851.0	2698.0	2491.0
15 - HS	2925.7	2905.0	1766.6	3008.5
15 - GS	1207.6	1290.4		
16 - HS	1249.0	1062.7	1559.5	1704.4
17 - HS	3008.5	4146.9	1766.5	2780.8
18 - HS	1000.6	1600.9	441.7	793.6
19 - HS	855.7	648.7	752.2	234.7
20 - HS	89.1	57.1	68.4	85.3

TABLE 3

Pb SOIL SAMPLES
GBC AREA
(μg of Pb/g of Soil)

SAMPLE NUMBER	1	2	3	4
1 - PS	42.0	45.8	32.6	43.9
2 - SS	45.8	21.3	42.0	49.6
3 - PS	49.6	51.4	62.7	64.6
4 - SS	117.3	258.4	53.3	11.9
5 - DS	87.2	160.6	136.1	192.6
6 - DS	51.4	154.9	203.9	198.2
7 - DS	74.0	139.9	79.7	115.4
8 - PS	30.7	23.2	75.9	57.1
9 - PS	122.9	49.6	53.3	185.0
10 - PS	79.7	62.7	75.9	100.4
11 - HS	136.1	793.6	138.0	335.6
12 - HS	62.7	121.1	89.1	79.7
12 - GS	139.9	102.3		
13 - HS	102.3	83.4	106.0	83.4
14 - HS	75.9	81.6	64.6	68.4
15 - HS	1042.0	81.6	160.6	94.7
16 - HS	91.0	164.4	121.1	117.3
17 - HS	100.4	151.2	139.9	119.2
18 - HS	752.2	179.4	66.5	77.8
18 - GS	234.0	177.5		
19 - HS	102.3	94.7	87.2	162.5
20 - HS	138.0	128.6	147.4	74.0

TABLE 4

Pb SOIL SAMPLES
REF AREA
(μg of Pb/g of Soil)

SAMPLE NUMBER	1	2	3	4
1 - PS	28.9	30.7	32.6	43.9
2 - SS	49.6	21.3	25.1	30.7
3 - PS	75.9	40.1	47.7	47.7
11 - HS	25.1	45.8	27.0	27.0
11 - GS	38.3	28.9		
12 - HS	27.0	27.0	21.3	23.2
13 - HS	32.6	30.7	27.0	32.6
14 - HS	25.1	19.4	17.6	13.8
15 - HS	21.3	19.4	21.3	23.2
16 - HS	66.5	117.3	94.7	10.0
17 - HS	77.8	92.8	64.6	60.8
18 - HS	36.4	38.3	74.0	38.3
19 - HS	15.7	27.0	23.2	25.1
20 - HS	38.3	21.3	4.4	275.4

TABLE 5

Cd SOIL SAMPLES
RSR Area
(µg of Cd/g of Soil)

SAMPLE NUMBER	1	2	3	4	
1 - PS	0.686	0.568	1.023	0.795	— 1
2 - PS	1.477	1.136	1.264	0.909	2
3 - PS	1.591	1.023	0.909	1.023	3
4 - SS	1.136	1.364	0.455	0.568	4
5 - PS	6.477	1.136	0.795	1.932	12
6 - DS	2.614	1.136	1.364	1.932	5
7 - PS	1.264	1.250	1.023	1.250	6
8 - DS	0.7950	0.682	0.568	0.795	7
9 - SS	0.227	0.795	0.455	0.227	8
10 - SS	2.727	0.455	2.159	2.273	9
11 - HS	2.727	2.614	2.386	2.159	10
12 - HS	2.955	4.432	1.477	1.591	11
13 - HS	2.727	6.477	2.614	7.955	X
14 - HS	1.136	2.841	24.205	9.432	X
15 - HS	0.114	1.250	1.705	0.795	13
16 - HS	2.159	2.386	3.182	2.614	14
17 - HS	0.568	0.455	0.455	1.705	15
18 - HS	1.477	1.136	1.364	1.705	16
19 - HS	1.932	1.705	1.591	1.591	17
20 - HS	1.364	1.477	1.932	1.932	18

TABLE 6

Cd SOIL SAMPLES
DMC AREA
(µg of Cd/g of Soil)

SAMPLE NUMBER	1	2	3	4
1 - PS	1.250	1.477	0.682	0.795
2 - PS	1.818	2.273	2.500	2.841
3 - PS	1.477	2.614	1.818	2.159
4 - PS	1.136	1.250	1.250	1.364
5 - PS	0.227	0.000	0.000	0.000
6 - PS	0.568	0.682	1.136	1.250
7 - SS	0.227	0.000	0.000	0.000
8 - PS	0.000	0.114	2.273	1.705
9 - SS	0.568	0.455	0.795	1.705
10 - PS	0.227	0.227	0.341	2.273
11 - HS	1.136	1.023	2.519	3.295
12 - HS	1.705	1.705	2.159	2.159
12 - GS	1.818	1.250		
13 - HS	0.144	0.568	2.159	1.364
14 - HS	6.477	5.455	3.636	2.841
15 - HS	1.136	1.250	1.250	2.955
15 - GS	1.023	0.568		
16 - HS	0.795	0.909	1.705	1.818
17 - HS	2.614	3.068	1.250	2.386
18 - HS	2.273	4.659	0.909	1.364
19 - HS	2.045	1.023	1.250	0.909
20 - HS	0.909	1.023	2.841	1.705

TABLE 7

Cd SOIL SAMPLES
GBC AREA
(μg of Cd/g of Soil)

SAMPLE NUMBER	1	2	3	4
1 - PS	0.909	0.795	1.023	1.136
2 - SS	0.000	0.114	0.114	1.136
3 - PS	0.568	1.477	0.114	0.114
4 - SS	0.114	0.909	0.114	0.455
5 - DS	0.341	0.455	0.455	0.227
6 - DS	0.114	0.682	0.795	1.023
7 - DS	0.909	0.795	0.341	0.227
8 - PS	0.341	0.000	1.023	0.000
9 - PS	0.114	0.114	0.227	0.091
10 - PS	0.114	0.227	0.682	0.341
11 - HS	0.568	4.432	0.682	1.591
12 - HS	0.227	0.341	0.455	0.455
12 - GS	1.591	0.795		
13 - HS	0.000	0.455	0.455	0.682
14 - HS	0.114	0.227	0.114	0.227
15 - HS	2.273	0.227	0.114	0.227
16 - HS	0.114	0.455	0.569	0.455
17 - HS	0.227	0.227	0.114	0.455
18 - HS	2.273	0.682	0.227	0.341
18 - GS	1.136	0.909		
19 - HS	0.227	0.114	0.455	2.159
20 - HS	0.795	0.909	0.057	0.455

TABLE 8

Cd SOIL SAMPLES
REF AREA
(μg of Cd/g of Soil)

SAMPLE NUMBER	1	2	3	4
1 - PS	0.341	0.114	0.114	0.227
2 - SS	0.114	0.227	0.114	0.114
3 - PS	0.114	0.227	0.114	0.114
11 - HS	0.568	0.341	0.114	0.341
11 - GS	0.341	0.341		
12 - HS	0.227	0.227	0.341	0.114
13 - HS	0.341	0.114	0.114	0.227
14 - HS	0.455	0.114	0.227	0.227
15 - HS	0.227	0.114	0.114	0.000
16 - HS	0.227	0.341	0.227	0.341
17 - HS	0.227	0.341	0.560	0.455
18 - HS	0.227	0.227	0.682	0.114
19 - HS	0.341	0.455	0.227	0.455
20 - HS	0.114	0.114	0.227	0.455

TABLE 9

Pb DUST TRAY SAMPLES
(28 DAY EXPOSURE)

SAMPLE NUMBER	REF AREA ($\frac{ng}{cm^2}$)	GBC AREA ($\frac{ng}{cm^2}$)	RSR AREA ($\frac{ng}{cm^2}$)	DMC AREA ($\frac{ng}{cm^2}$)
11 - 1	25.7	17.9	71.4	26.3
12 - 1	6.4	5.0	45.2	61.0
13 - 1	7.6	7.1	44.5	8.8
14 - 1	16.7	25.7	28.8	48.1
15 - 1	6.9	12.8	10.8	45.2
16 - 1	11.2	11.0	51.1	65.9
17 - 1	10.2	13.6	10.9	138.0
18 - 1	8.9	29.7	18.8	12.5
19 - 1	5.7	4.5	36.3	32.2
20 - 1	6.9	20.0	46.7	6.5

TABLE 10

Cd DUST TRAY SAMPLES
(28 DAY EXPOSURE)

SAMPLE NUMBER	REF AREA $(\frac{ng}{cm^2})$	GBC AREA $(\frac{ng}{cm^2})$	RSR AREA $(\frac{ng}{cm^2})$	DMC AREA $(\frac{ng}{cm^2})$
11 - 1	1.53	1.07	1.00	1.30
12 - 1	1.92	0.54	0.77	2.07
13 - 1	1.46	0.84	1.84	1.53
14 - 1	1.46	0.77	1.76	1.76
15 - 1	1.30	1.15	0.77	1.84
16 - 1	1.38	0.84	1.76	2.07
17 - 1	0.84	1.38	1.00	2.23
18 - 1	1.30	1.23	1.53	1.69
19 - 1	1.99	1.46	1.15	1.07
20 - 1	1.00	2.92	2.15	1.53

TABLE 11

Pb DUST WIPE SAMPLES
(100 sq cm AREA)

REFRIGERATOR SAMPLE NUMBER	REF AREA ($\frac{\mu g}{cm^2}$)	GBC AREA ($\frac{\mu g}{cm^2}$)	RSR AREA ($\frac{\mu g}{cm^2}$)	DMC AREA ($\frac{\mu g}{cm^2}$)
11 - 1	0.26	0.18	1.58	0.37
12 - 1	0.12	0.72	1.56	0.33
13 - 1	0.13	0.67	0.41	0.47
14 - 1	0.23	0.22	0.23	2.77
15 - 1	0.51	0.24	0.40	0.26
16 - 1	0.39	0.18	1.86	0.54
17 - 1	0.37	0.42	0.63	1.79
18 - 1	0.18	0.26	0.24	0.31
19 - 1	0.38	0.42	0.86	0.78
20 - 1	0.16	1.11	0.90	0.50
WINDOW SILL SAMPLE NUMBER				
11 - 2	0.16	0.14	3.26	0.76
12 - 2	0.17	5.97	0.61	0.40
13 - 2	0.18	1.33	1.86	0.23
14 - 2	0.27	0.46	0.37	9.61
15 - 2	0.39	0.34	1.35	0.43
16 - 2	1.37	0.53	1.24	0.72
17 - 2	0.63	7.54	0.19	1.89
18 - 2	0.39	0.23	1.24	6.04
19 - 2	0.20	0.41	11.05	6.83
20 - 2	0.36	0.63	1.61	0.96

TABLE 12

Cd DUST WIPE SAMPLES
(100 sq cm AREA)

REFRIGERATOR SAMPLE NUMBER	REF AREA ($\frac{ng}{cm^2}$)	GBC AREA ($\frac{ng}{cm^2}$)	RSR AREA ($\frac{ng}{cm^2}$)	DMC AREA ($\frac{ng}{cm^2}$)
11 - 1	10.0	10.0	46.7	18.7
12 - 1	6.5	33.7	13.6	10.0
13 - 1	5.7	45.9	12.9	13.6
14 - 1	9.3	7.2	8.6	48.8
15 - 1	14.4	7.9	13.6	10.0
16 - 1	25.8	12.2	12.2	8.6
17 - 1	10.8	16.5	7.9	9.3
18 - 1	7.2	12.2	5.7	9.3
19 - 1	32.3	15.1	57.4	38.0
20 - 1	13.6	7.9	8.6	29.4
WINDOW SILL SAMPLE NUMBER				
11 - 2	6.5	7.9	46.7	12.2
12 - 2	61.0	37.3	11.5	11.5
13 - 2	10.0	59.6	12.2	15.8
14 - 2	12.9	18.7	11.5	12.9
15 - 2	13.6	25.8	7.9	9.3
16 - 2	102.6	25.8	14.4	10.0
17 - 2	15.1	19.4	5.7	20.1
18 - 2	7.2	11.5	7.2	13.6
19 - 2	7.9	17.2	17.9	68.2
20 - 2	7.9	7.9	10.0	7.2

TABLE 13

Pb HOUSE WATER SAMPLES

COLD WATER SAMPLE	REF AREA (PPb)	GBC AREA (PPb)	RSR AREA (PPb)	DMC AREA (PPb)
11 - 1	2.27	1.04	1.24	1.59
12 - 1	1.23	1.05	0.51	0.65
13 - 1	1.65	0.99	0.61	1.74
14 - 1	2.06	1.13	0.51	0.88
15 - 1	0.41	0.66	0.51	0.65
16 - 1	0.39	1.76	0.40	1.83
17 - 1	0.39	0.93	0.51	1.00
18 - 1	0.50	1.32	0.51	0.53
18 - 2		0.58		
19 - 1	0.60	1.26	0.92	2.54
19 - 2				1.00
20 - 1	0.82	1.04	0.51	1.24

HOT WATER SAMPLE				
11 - 2	0.82	2.05	0.61	0.65
12 - 2	1.23	2.48	0.30	1.55
13 - 2	2.89	3.14	1.34	2.71
14 - 2	1.44	2.97	0.82	1.00
15 - 2	7.28	0.82	0.72	1.83
16 - 2	1.67	2.54	0.51	1.24
17 - 2	0.82	3.14	1.13	1.47
18 - 2	2.81		0.19	1.97
19 - 2	2.21	0.55	0.51	
20 - 2	0.39	2.07	0.40	0.88

TABLE 14

Cd HOUSE WATER SAMPLES

COLD WATER SAMPLE	REF AREA (PPb)	GBC AREA (PPb)	RSR AREA (PPb)	DMC AREA (PPb)
11 - 1	0.00	0.06	0.05	0.00
12 - 1	0.00	0.07	0.06	0.00
13 - 1	0.00	0.08	0.05	0.02
14 - 1	0.05	0.09	0.15	0.05
15 - 1	0.03	0.06	0.02	0.00
16 - 1	0.00	0.11	0.12	0.04
17 - 1	0.00	0.09	0.17	0.04
18 - 1	0.00	0.06	0.10	0.04
18 - 2		0.09		
19 - 1	0.00	0.07	0.30	0.10
19 - 2				0.02
20 - 1	0.03	0.05	0.04	0.02

HOT WATER SAMPLE				
11 - 2	0.00	0.06	0.10	0.09
12 - 2	0.00	0.07	0.02	0.06
13 - 2	0.00	0.08	0.08	0.09
14 - 2	0.05	0.09	0.07	0.06
15 - 2	0.03	0.08	0.10	0.09
16 - 2	0.00	0.11	0.27	0.04
17 - 2	0.00	0.08	0.07	0.06
18 - 2	0.00		0.09	0.06
19 - 2	0.00	0.07	0.16	
20 - 2	0.05	0.06	0.03	0.05

TABLE 15

CADMIUM DATA SUMMARY

HOUSE TAP WATER SAMPLES (ppb)

	AREA			
	REF	GBC	RSR	DMC
$\bar{x}$	0.012	0.077	0.103	0.047
S.D.	0.019	0.016	0.074	0.030

DUSTFALL OVER A 28-DAY PERIOD (ng/cm²)

	AREA			
	REF	GBC	RSR	DMC
$\bar{x}$	1.40	1.22	1.37	1.71
S.D.	0.31	0.63	0.47	0.35

REFRIGERATOR TOP DUST WIPE SAMPLES (ng/cm²)

	AREA			
	REF	GBC	RSR	DMC
$\bar{x}$	13.56	16.86	18.72	19.57
S.D.	8.32	12.14	17.03	13.57

WINDOW SILL DUST SAMPLES (ng/cm²)

	AREA			
	REF	GBC	RSR	DMC
$\bar{x}$	24.47	23.11	14.50	18.08
S.D.	30.26	14.88	11.25	17.05

SOIL SAMPLES (ppm)

	AREA			
	REF	GBC	RSR	DMC
$\bar{x}$	0.25	0.58	2.05	1.50
S.D.	0.14	0.66	2.95	1.18

TABLE 16

LEAD DATA SUMMARY

HOUSE TAP WATER SAMPLES (ppb)

AREA

	REF	GBC	RSR	DMC
$\bar{x}$	1.59	1.58	0.64	1.35
S.D.	1.53	0.85	0.30	0.61

DUSTFALL OVER A 28-DAY PERIOD (ng/cm^2)

AREA

	REF	GBC	RSR	DMC
$\bar{x}$	10.62	14.73	36.45	44.45
S.D.	5.89	8.12	18.34	37.03

REFRIGERATOR TOP DUST WIPE SAMPLES (μg/cm^2)

AREA

	REF	GBC	RSR	DMC
$\bar{x}$	0.273	0.442	0.867	0.812
S.D.	0.126	0.289	0.571	0.779

WINDOW SILL DUST WIPE SAMPLES (μg/cm^2)

AREA

	REF	GBC	RSR	DMC
$\bar{x}$	0.412	1.758	2.278	2.787
S.D.	0.348	6.46	3.04	3.22

SOIL SAMPLES (ppm)

AREA

	REF	GBC	RSR	DMC
$\bar{x}$	54.0	131.3	494.1	1253.7
S.D.	41.7	155.2	509.7	3869.3

$\bar{x}$ = Arithmetic mean

S.D. = Standard deviation

CONCLUSIONS

Cadmium in House Tap Water Samples

Cadmium levels in house tap water was low, averaging over the eighty samples collected about 0.06 parts per billion (ppb) cadmium. There were no individual samples which showed concentrations high enough to generate any concern for exposure to cadmium from tap water.

There appeared to be a possibility that cadmium levels in tap water were higher in the GBC and RSR areas than in the control area (0.077 and 0.103 ppb vs. 0.012 ppb). However, since all samples were from public water supplies, it would not seem likely that any variation would be due to the proximity of lead handling operations.

Cadmium Dustfall Over a 30-Day Period

Cadmium levels in dustfall were quite low, averaging over the entire study 1.43 nanograms of cadmium per square centimeter (ng/cm^2). Levels in the DMC area, on average, were approximately 20 percent above the other areas.

Cadmium Dust Wipe Samples

Cadmium levels found in wiped-up dust from the top of refrigerators and from window sills were low. The average for refrigerator tops was 17.18 (ng/cm^2) and for window sills averaged 20.04 (ng/cm^2).

Refrigerator top samples averaged lowest in the REF area and increased from 13.56 (ng/cm^2) in this area to 16.86, 18.72, and 19.57 (ng/cm^2) in the GBC, RSR, DMC areas, respectively. Conversely, window sill wipes were highest in the REF area and decreased from 24.47 (ng/cm^2) in this area to 23.11, 14.50, and 18.08 (ng/cm^2) in the GBC, RSR and DMC areas, respectively.

Cadmium in Soil

The average concentration of cadmium in soil in the REF area was 0.25 micrograms of cadmium per gram of soil (µg/g). Cadmium in soil in the other study areas averaged: GBC, 0,58 (µg/g); RSR, 2.05 (µg/g); and DMC, 1.50 (µg/g). The highest cadmium concentration sampled was 24.2 (µg/g) in sample RSR-14-HS-3, a soil sample taken from a house yard.

Lead in House Tap Water Samples

Lead in house tap water samples was low. On average, lead concentrations from hot water taps was slightly higher than cold water taps. For the entire study, lead in cold tap water averaged 1.01 part per billion (ppb) and in hot tap water averaged 1.57 ppb. While there were differences in average concentrations of lead in tap water from the various areas, none of the differences would likely be due to lead handling operations in the area.

Lead Dustfall Over a 30-Day Period

Compared to the REF area, lead in dustfall was elevated on average 38.7% (GBC); 243.2% (RSR); and 319.0% (DMC). The REF area averaged 10.62 nanograms of lead dustfall per square centimeter (ng/cm^2) over the 30-day period, while the other areas averaged: 14.73 ng/cm^2 (GBC); 36.45 ng/cm^2 (RSR) and 44.45 ng/cm^2 (DMC).

The average lead dustfall over the entire study area was 26.56 nanograms per square centimeter per month. This is equal to 0.2656 milligrams per square meter per month (mg/m^2. mo.). This value compares with outdoor dustfall values of 5.24 mg/m^2. mo. in residential areas, 9.80 mg/m^2. mo. in commercial areas and 12.78 mg/m^2. mo. in industrial areas reported in Air

Quality Criteria for Lead, EPA-600/8-77-017, December, 1977. The Southwest Research Institute (SRI) study, Epidemiologic Study of the Effect of Automobile Traffic on Blood Lead Levels, EPA Contract Number 68-02-2227, June, 1978, examined the relationship between indoor and outdoor lead concentrations and reported the ratio 1/10 for this condition. Thus, the values sampled in this study appear to be of appropriate order of magnitude.

Lead Dust Wipe Samples

Refrigerator top lead concentrations averaged 0.273 micrograms per square centimeter (μg/cm²) in the REF area, and increasing respectively as: 0.442 μg/cm² (GBC), 0.867 μg/cm² (RSR), and 0.812 μg/cm² (DMC). This general pattern was repeated for the window sill wipes, with the REF area averaging 0.412 μg/cm², and 1.76 μg/cm² (GBC), 2.28 μg/cm² (RSR), and 2.79 μg/cm²(DMC).

There were some values of window sill dust wipes which were considerably higher than most. A value of 11.05 μg/cm² (RSR-19-2) and values of 9.61 μg/cm² (DMC-14-2), 6.04 μg/cm² (DMC-18-2) and 6.83 μg/cm² (DMC-19-2) increased the average values in these areas quite significantly. It would be interesting to determine if these window sills were painted with lead paint.

Lead in Soil

Soil lead in the REF area averaged 41.68 ppm, while soil leads in the other areas averaged 131.31 ppm (GBC, 494.09 ppm (RSR), and 1253.66 ppm (DMC).

No samples in the REF area contained 1000 ppm lead, while one sample (GBC-15-HS-1) in the GBC area contained greater than 1000 ppm lead. Seven sample areas in the RSR area contained lead concentrations greater than 1000 ppm. RSR-5-PS, RSR-6-DS, RSR-11-HS, RSR-12-HS, RSR-13-HS, RSR-19-HS, and RSR-20-HS. While these samples contained greater than 1000 ppm lead, all except RSR-6-DS were soil samples from house yards. RSR-6-DS had one soil sample with a concentration of 1683.7 ppm. Since this sample was from a day care center playground, it may be of value to verify this concentration and better define its area of significance if found to be valid.

There were also seven areas with greater than 1000 ppm found in the DMC area. One of these areas, DMC-14-HS had extremely high concentrations (25,503 ppm). Those areas which contained greater than 1000 ppm lead were: DMC-1-PS, DMC -11-HS, DMC-14-HS, DMC-15-HS, DMC-16-HS, DMC-17-HS, and DMC-18-HS. Since DMC-1-PS is a playground soil sample, it would be of interest to verify this concentration of greater than 1000 ppm. Additionally, the DMC-15-GS garden soil samples had greater than 1000 ppm lead and could result in elevated lead concentrations in food grown in this garden.

It would be interesting to better define the cause of the very high soil lead values found in DMC-14-HS. Additionally, if children play in this yard, perhaps it would be appropriate for their parents to be advised of the advantages of screening for elevated body burden of lead in the children.

APPENDIX A

SITE LOCATION

DMC-1	Playground, Corner of Cadillac and Pontiac
DMC-2	Playground, Corner of Childes and Avenue H
DMC-3	S. Moore Park on Rockefeller Blvd.
DMC-4	N. Moore Park on Ventura Drive
DMC-5	Playground, Corner of Cliff and Sabine
DMC-6	Playground, Corner of South and Gould
DMC-7	St. Phillips School on Pennsylvania
DMC-8	Wonder View Park on Kiest Blvd.
DMC-9	William Brown Miller Elementary School on Bonnie View
DMC-10	Sargent Park on Sargent Road
DMC-11	420 N. Dently
DMC-12	2215 Rockerfeller
DMC-13	427 DuBois
DMC-14	1023 Pontiac
DMC-15	713 Buick
DMC-16	720 Lasel
DMC-17	2963 Gloyd
DMC-18	2608 Guold
DMC-19	1312 Pennsylvania
DMC-20	2424 Magnavista
GBC-1	Weichsel Park on Denton Drive
GBC-2	Playground on Inwood
GBC-3	Playground on Corner of Cedar Springs and Hedgerow
GBC-4	School Playground, Corner of Tyree and Thedford
GBC-5	Day Care Center, 6544 Kenwell Drive

GBC-6	Brentwood Day Care Center on Corner of Oriole and Inwood
GBC-7	Shaptaw Child Care Center on Inwood
GBC-8	Maple Park, Corner of Hawthorne and Maple
GBC-9	Playground, Corner of Hines Blvd and Anson
GBC-10	Playground, Corner of Canada Drive and Winnetka
GBC-11	6519 Prosper Street
GBC-12	5804 Nash
GBC-13	3702 W. Fair Fax
GBC-14	3319 Hedgerow
GBC-15	5423 Stoneleigh
GBC-16	5123 Denton Drive
GBC-17	5312 Parkland
GBC-18	2710 Kimsey
GBC-19	2603 Bomar
GBC-20	7006 Concord
RSR-1	Playground, Corner of Norma and Doug Drive
RSR-2	Playground on Bickers near Pointer intersection
RSR-3	Playground on Bernal near Ladale intersection
RSR-4	Amelia Elementary School on Westmoreland
RSR-5	Playground, corner of Magdeline and Esmaida
RSR-6	Tabernacle Day Care Center, 3403 McBroom
RSR-7	Playground on Canada Road, West of Pointer
RSR-8	West Dallas Development Center, 2650 Canada Drive
RSR-9	Carver Elementary School, Corner of Holystone and Brickers
RSR-10	Jose Antonio Navarro School on Kingsbridge
RSR-11	3402 Nomas

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EPA Environmental News

EDDIE LEE
(214) 767-2630

FOR IMMEDIATE RELEASE JUNE 1, 1981

The U. S. Environmental Protection Agency (EPA) today released an EPA funded study, investigating lead and cadmium levels near industrial sources in Dallas. The study shows elevated lead levels in soil near three lead and battery plants.

Samples of lead and cadmium were taken from residential neighborhoods and schools within two miles of Dixie Metals Company, NL Industries, RSR Corporation and General Battery Company and within a reference area remote from known sources of lead and cadmium. Analysis of indoor dust and tap water samples revealed no significant problems with respect to lead or cadmium. Analysis of soil samples indicated that, although cadmium levels were not significantly high, lead levels did show marked elevation in the test areas.

The study shows soil lead levels in the test areas ranging from an average of 131 parts per million (ppm) to 1,253 ppm. The average soil lead level in the reference area was 54 ppm. A 1978 study of lead levels in soil near major traffic arteries in Dallas indicated an average of 128 ppm.

EPA has begun meeting with State and local officials and representatives of the companies to determine the existence and the scope of any environmental problem and to discuss possible corrective actions. EPA has no evidence of medical injury to residents of the test area.

(more)

Dallas city health officials are reviewing historical blood lead information collected during the past few years through the Early Periodic Screening, Diagnosis and Treatment Program and the Children and Youth Clinic in West Dallas to determine if there is any correlation between the elevated soil lead levels and any increased blood lead levels of children residing in the study areas. No evidence, to date, of any lead concentration justifying medical intervention has been found. However, a number of children, not necessarily living in the areas presently being studied, have exhibited lead concentrations above what is considered normal levels.

Frances E. Phillips, Acting Regional Administrator of EPA for the five state area including Texas, says, "We anticipate the continued cooperation of the companies, and commend RSR Corporation for taking the lead in the voluntary commitments made by the companies involved."

In response to the study, RSR will develop, with EPA, an expanded soil sampling program to determine the extent and magnitude of the environmental problem around its facilities, if any; and support additional biological monitoring to the extent deemed necessary. In addition, RSR will take remedial measures to reduce the soil lead levels in high risk areas such as playgrounds, day care centers, and school yards, where lead concentrations are over 1,000 ppm.

Dixie Metals Company has indicated a willingness to cooperate in corrective measures where needed, including an expanded soil sampling program and the cleanup of soil areas if necessary. They have also indicated a willingness to financially support needed additional blood sampling.

The boundaries of the two areas of further study are as follows: The first test area, located in south Dallas, within a two mile radius downwind of Dixie Metals and NL Industries facilities is divided into two sections. The first section is located on the north flood plain of the Trinity River bounded by Lamar on the south, I-45 on the east, AT&SF Railroad on the west and by Ervay and Forest on the north. The second section is located on the south flood plain of the Trinity River bounded by the Trinity on the north, Sargent Drive on the east, Cedar Crest Blvd. on the west and on the south by Kiest, Rector and Nolen.

The second test area, in the vicinity of RSR, located in west Dallas, is bounded by the Trinity River on the north, Norwich on the west, T&P Railroad on the south and Kingsbridge on the east.

To assist those living in the test areas who are concerned about possible lead effects, EPA and Dallas health officials advise them to contact their family doctor or Dr. Lowell Berry, Dallas City Health Department, at (214) 670-6634, if any symptoms appear. According to Dr. Norman Dyer, Chief of EPA's Hazardous Materials Branch, the most common symptoms of lead poisoning are anemia, severe intestinal cramps, paralysis of nerves (particularly in the arms and legs), loss of appetite and fatigue. Immediate remedies are available and individuals can be detoxified with proper medical care.

Persons wanting additional information about the study should contact Dr. Norman E. Dyer, EPA, at (214) 767-2734.

Mr. **Levitas**. The gentleman from New York.

Mr. **Molinari**. Before we leave that question, doctor, we did notice the similarity in the handwriting. I'm no expert in the handwriting of Mr. Rosius and the author of some of the comments. So I would appreciate it if you would still comply with the request that we made before.

Dr. **Hernandez**. Absolutely.

Mr. **Molinari**. OK.

I just want to get back to the questions about political backgrounds and try to clear the air, because the information that I received this morning seems to be in conflict with information that the minority staff has received.

Now, I am going to try to be more specific, Dr. Hernandez, as you requested.

Numerous allegations have been made with respect to lists of the political backgrounds or political leanings of people, maybe philosophical leanings, at the Environmental Protection Agency. I am asking you now, are you aware of any such lists?

Dr. **Hernandez**. I have never seen a list other than the one that came in the last couple of weeks that referred to EPA employees specifically and their political leaning that was the one that I think the list that has been circulated with some 90 names on it. By and large, all those people are outside of EPA, not full-time employees, but are what you call consultants or some such thing.

Mr. **Molinari**. Were you aware of any lists containing any names of people with political affiliations who may have been consultants to EPA?

Dr. **Hernandez**. I believe that that one list that has been around and there may be other versions of that list around.

Mr. **Molinari**. When did you first become aware that there was such a list?

Dr. **Hernandez**. About 2 weeks ago that I heard about it, that I knew that it existed as a list.

Mr. **Molinari**. Maybe you might have heard something about individuals and not specifically a list, so let's change the line of questioning. Were you aware that questions had been raised about either employees or consultants to the Environmental Protection Agency specifically with respect to their political background or their political philosophy?

Dr. **Hernandez**. Well. People who should be fired or not hired because of that particular reason, as either Federal employees or as consultants?

Mr. **Molinar**. Well, I'm asking a kind of broad question.

Dr. **Hernandez**. Yes.

Mr. **Molinari**. I don't know whether they should have been fired or not.

Dr. **Hernandez**. That was the intent of the list. That was the intent of the list.

Mr. **Molinari**. That was the intent of the list?

Dr. **Hernandez**. That's what I was saying. You're saying that there—was there any idea that there was a list of people who shouldn't be hired?

Mr. **Molinari**. No, no. No, I'm not really asking that.

Dr. **Hernandez**. Maybe I'm wrong.

Mr. MOLINARI. They may have been consultants or they may have been employees. It may not have been a question of hiring or firing, but a question of, watching these people.

Dr. HERNANDEZ. No; I don't know of any list like that.

Mr. MOLINARI. Now, when there were background checks made of anybody that was scheduled to be hired, or to be employed as consultants, was there anybody to your knowledge who made any political assessments on that person before they received a consulting contract or employment at EPA?

Dr. HERNANDEZ. Not specifically with respect to consultants, but in any administration you have about three kinds of positions where this comes into account. Those are the schedule C's, and there are a limited number of those. The next one is something called "administratively determined," and I'm not sure what the formula is on that, and then the third one is noncareer senior executive service people. Again, there is some kind of a limitation on those people, and that list group is an example. The regional administrators of EPA's regions are by and large noncareer SES people, senior executive service people, who it is not inappropriate to say, because they are members of this administration, "What are your basic political philosophies on how you should deal with this sort of thing? And those kinds of people, as you go through the process of recruiting people, I believe that that—

Mr. MOLINARI. I will be glad to yield to the Chairman.

Mr. LEVITAS. Would the gentleman yield at that point.

What about persons being considered for appointment to science advisory groups?

Dr. HERNANDEZ. OK.

Mr. LEVITAS. Were there any lists that assessed the political or philosophical orientation of persons who either had been serving or were being considered for future service?

Dr. HERNANDEZ. The only one I saw was the one that came out recently that characterized people.

You have to remember that we got lists from all kinds of people sent to us. And we went out into the world and asked them to send us lists, and some of those may have had characterizations on there; you know, somebody might have said, "boy, this guy is a great Republican" or "some crazy." You can't control that. They were written to you, and they came in the door with everything else that we got.

Mr. LEVITAS. Have you seen such lists?

Dr. HERNANDEZ. Not as lists per se. When you get—those are individual kinds of things. Somebody sent us a name or something like that. And they derive from all kinds of places.

Mr. LEVITAS. Do you have in your possession any such characterization? I am talking now about scientists.

Dr. HERNANDEZ. In terms of individuals or are you talking about a compiled list that has characterizations? Because there is a difference.

Mr. LEVITAS. A document that contains the name of more than one person and has a characterization about their political or philosophical——

Dr. HERNANDEZ. Yes.

I don't think so at all that that's in there. Let me tell you the kinds of places we got more than one name from——

Mr. **Levitas**. I am not finished with my question——

Dr. **Hernandez**. Sure.

Mr. **Levitas** [continuing]. If I may.

Dr. **Hernandez**. Yes.

Mr. **Levitas**. My question really pertains—and the reason I'm targeting on scientists is—what would be the relevancy of the political or philosophical orientation of a scientist, No. 1; and No. 2, do you know of any such scientists who had been serving on some of these boards during prior administrations who were after such information, no longer invited to participate?

Dr. **Hernandez**. And the answer to that is, for the Science Advisory Board, inappropriate that you know what somebody's political registration or political philosophy per se is. You should be a physicist first or a chemist first or a stream biologist, and your competency has to be based on your track record out there in your science; and when you are making choices, you may say, how does this guy get along with other folks; but that's a different kind of thing; that's ability to work with committee members and things and has nothing to do with his philosophical viewpoint, as you have indicated. And I think the second part of your question was, was there lists of people that served prior administrations on these boards where indications were about their philosophy. The only one I have seen is that one that has been circulated recently.

Mr. **Molinari**. Has there been any assessments made in the hiring of consultants, outside consultants, based on their published papers and their attitudes on some of the environmental issues that are confronting EPA?

Dr. **Hernandez**. We have to separate that just a little bit into two questions.

Mr. **Molinari**. OK.

Dr. **Hernandez**. One, their published papers, yes, I read abstracts of what people have to write, and try to make those judgments on whether they would be good contributors or not based on those papers in a scientific kind of sense. I think the other part of the question, do you try to gain any kind of philosophical thing out there, and I think the answer is no.

Mr. **Molinari**. I take it you have quite a bit of say, and have in the past, on the hiring of those who would fall in the category of scientists because you are a scientist yourself and were previously Deputy Administrator.

When your announcement appeared in the paper, particularly the New York Times, you might have seen a reference that I made to acid rain and how we disagree on that aspect. What I am interested in really is, would you hire somebody whose views scientifically might differ radically from yours?

Dr. **Hernandez**. Certainly, and, you know, I'm going to say we have got a number of people on the SAB Boards and things that believe that you—and this is——

Mr. **Molinari**. Let's zero in on consultants.

Dr. **Hernandez**. Let me give an example. Yes, consultants. Those people are consultants.

Mr. **Molinari**. OK.

Dr. **Hernandez**. Where they believe you have to have this much more information before you can make this kind of a determination and I think you could probably do it with lesser information; and we have those people on and we have wonderful disagreements and debates on those issues.

Mr. **Molinari**. All right. I'm pleased to hear that.

One last question.

Dr. **Hernandez**. All right.

Mr. **Molinari**. I am going to repeat the question which I asked earlier about Mr. Herbolsheimer, and the alleged difficulties that he was having with Mr. Chip Wood. The information again, as I said earlier, was that Wood had questioned Herbolsheimer about his political affiliations, and then Herbolsheimer, who was upset at such questions, came back to you and related the conversation.

Dr. **Hernandez**. Let me see if I can straighten out more or less what happened there.

Mr. Herbolsheimer had all of these lists, a large number of committees, some science advisory board, some drinking water advisory committee, some administrator's toxic substances, lots of names, some of them appropriate here, not appropriate there, based on background, some scientists, some nonscientists.

Mr. Wood who—as I understood his position at the agency, was to look at schedule C's, administratively determined people and noncareer SES's during that initial kind of process when we were getting under way and filling a few of those spots—had asked Mr. Herbolsheimer to look at—determine some of the appropriateness of putting people on some of these Boards, and some of those—what Mr. Herbolsheimer did do, and I don't want to characterize at all, because I don't know all, but I'll try to give it to you as best I understand it—was to call a congressional office and say, "Mr. Molinari, have you ever heard of this man? We're thinking about appointing him to this group or this group; do you have any problems?" And that was—occurred on some of these committee people in the other.

I think there was about a 3 month period when there was some involvement of Mr. Wood in that process. Mr. Herbolsheimer came and talked to me about it and the difficulty of doing this. And when I found it applied to Science Advisory Board people, I then went and talked to the Administrator about that issue, and sometime after Christmas when she returned from vacation, we resolved that issue totally and got Mr. Wood away from that process.

Mr. **Molinari**. Your testimony now is a little different than that of this morning.

Dr. **Hernandez**. Well, I think that I was misunderstanding it. It sounded like what you were saying was that Mr. Herbolsheimer's affiliations, political affiliations were being questioned.

Mr. **Molinari**. I was going to lead from there into the fact that he was then asked about others.

Dr. **Hernandez**. Yes.

Mr. **Molinari**. His own affiliations and others.

Dr. **Hernandez**. I don't think his own was ever challenged, that I know of.

Mr. **Molinari**. But others were?

Dr. HERNANDEZ. At least, would you check on these guys. The process that Herbolsheimer tried was the one of contacting Congressmen, which is not an inappropriate thing to notify a Congressman that some one may be on a committee.

Mr. MOLINARI. Now, you did take this information and go to Mrs. Burford at the time and would you mind just telling us how that conversation went?

Dr. HERNANDEZ. It kind of was a two-staged one. The second one was a satisfactory process that we worked out where I would review written—you know, for these scientists that were going on these things—would review documents that they had written where I personally didn't know the individual as to his or her technical expertise. And by reading abstracts of articles, to ask him if they were in fact good scientists. Because otherwise you have to go—you know, we get a recommendation from somebody on an individual, which we got many, many I didn't know, it was difficult to determine whether or not in fact these people, other than what you can read on a vitae, what was their track record, and so you had to go read articles; and what I did is read abstracts of articles by these people.

We also did another thing on this point on the SAB. I went and talked to the executive committee of the Science Advisory Board which was—at that time we had made one replacement. All the rest were people who were there from the previous administration. What we ought to do by and large is review possible names with you. You all know these people much better than I do because I have a—I come out of one part of the sanitary engineering field and there's air pollution people and there's biologists and people who are specialists in other fields, and we want your nominations for names and this is the process we have used since then, by and large relying on the Science Advisory Board committees that were in existence to give us recommendations of names; but we had others, to try them on them and see if they sounded appropriate.

Mr. MOLINARI. Dr. Hernandez, when you went to Mrs. Burford, did you complain about the activities of Mr. Wood?

Dr. HERNANDEZ. Well, just that I felt it was inappropriate as a process or any kind of review on political or philosophical ideas of people who serve on the Science Advisory Board.

Mr. MOLINARI. So you did complain to her about this?

Dr. HERNANDEZ. Yes.

Mr. MOLINARI. Thank you very much.

Mr. LEVITAS. Thank you, Mr. Molinari.

Just one followup on that.

Do you know whether any scientists were purged from or not given consideration for appointments to Science Advisory Board because of their affiliations or their philosophy?

Dr. HERNANDEZ. No. I don't. That first year that we were there, we made one appointment to the Science Advisory Board. Everybody else remained exacly the same group we inherited. We did two things, though: We allowed appointments to expire and we did not reappoint anybody. There were a couple of reasons for doing that. The committees were quite large and we wanted them to come down to a size of about 10 to 12 rather than having as many as 18 on a committee, and so we made 1 appointment, and so that meant in that first year, about 6 appointments would lapse, so that we got down to the 10 or 12, and then as the next round of appointments came up, we have replaced them and we have reappointed some.

So that we have reappointed many people who were already there.

Mr. **Levitas.** Mr. Cannon, I'd like to ask you the same general question that Mr. Molinari asked Dr. Hernandez.

Were you aware, have you seen, or do you know the whereabouts of any lists of employees or consultants or others who were either employed by or being considered for employment by the Environmental Protection Agency that evaluated their political or philosophical attitudes?

Mr. **Cannon.** I have seen since I have been at the agency two lists. One list was the list that was just recently made public and I don't know the origin of that list, that had names and evaluations of people on the list.

I saw another list and I can't even really recall the date. It had a few names on it of people at the agency, some schedule C's from the previous administration, that—I just saw that list. I don't know—I have been—anything else I know is—would just have to be hearsay, you know, that I have heard lots of different things around here.

Mr. **Levitas.** This isn't a court of law. It is permissible for you to tell us the best information you've got. You seem like a very credible fellow. I believe you when you say that you heard it from somebody and I can evaluate it for what it is.

Mr. **Cannon.** At one point, Chip Wood did tell me that Bob Herbolsheimer had memoranda on different people. Now whether they were scientists or not scientists, I don't know. But I did know that there—I don't know this either—that Mr. Woods had said that their political affiliation was being looked at. And I have also been told that it is possible that Mr. Herbolsheimer may have such memoranda in his files.

Mr. **Levitas.** That Mr. Woods?

Mr. **Cannon.** Mr. Herbolsheimer.

Mr. **Levitas.** Mr. Herbolsheimer might have such lists in his files.

Is Mr. Herbolsheimer here today?

Mr. **Cannon.** He is not here today.

Mr. **Levitas.** If he has such lists in his files, of course, I would like to have copies of them, Dr. Hernandez.

Dr. **Hernandez.** We're making up a list for Congressman Scheuer of everything that we have with respect to the appointment of SAB and Mr. Herbolsheimer had for I believe the SAB, Science Advisory Board, on these lists. I think he has one which I have not seen, which I have not seen, never heard about until this folderol, which may be a slightly different version of the same list that has appeared, but I don't think it's substantively different. I think it is just a different version.

Mr. **Levitas.** I would like to have a copy of whatever list Dr. Herbolsheimer has.

Dr. **Hernandez.** Yes; and we will give you those—we have a summary of names we received and letters that we received from people. You know, when you ask, was there a list from other people, naturally we had so many from people, some of those may have characterizations. I just never looked through them and read them.

Mr. Levitas. All right.

One other question, Mr. Cannon. Where did you—you refer to two lists that you have seen.

When did you see the first list, if you can recall, and where did you see it?

Mr. Cannon. I can't remember when I saw it, sometime—it seems to me longer than a year ago, earlier in the administration, and I saw the list as I recall it in—where was I physically when I saw it?

Mr. Levitas. Yes. Were you in your office, in the cafeteria—there is no cafeteria—in your office——[Laughter.]

Where did you see it?

Mr. Cannon. I guess I was in the administrator's dining room.

Mr. Levitas. And was it just lying on the table, or did somebody show it to you or—

Mr. Cannon. No. I just—I can't remember who showed it to me. I just remember looking over someone's shoulder and seeing a list with five or six names on it. I don't even know what importance to attach to it. There was only a list of names and not characterizations, as in the other more massive list that I saw only just recently.

Mr. Levitas. And where was that and when did you—when was that and where did you see that?

Mr. Cannon. The longer list?

Mr. Levitas. Yes.

Mr. Cannon. I saw that during a break in our appropriations hearings just a few weeks ago in front of Mr. Boland. It happened to be the day it broke, and Lee Modiset, of our congressional liaison, had a copy of it, and he said, have you ever seen this, and that's the first time I had ever seen that kind of thing.

Dr. Hernandez. And I think all of us there before Mr. Boland said we had not seen that list.

Mr. Levitas. OK.

I have some more questions but I just wanted to clarify that part of it.

Dr. Rowland

Dr. Rowland. Thank you, Mr. Chairman.

I wanted to ask Mr. McPhee a couple of questions.

Mr. McPhee. Yes, sir.

Dr. Rowland. Mr. McPhee, you are from region V?

Mr. McPhee. I am.

Dr. Rowland. Are you familiar with the compilation, or involved in the compilation of this report that Dow Chemical Co. asked some part of it to be deleted?

Mr. McPhee. I was asked to review the report for legal considerations.

Dr. Rowland. Do you have any idea why Dow Chemical Co. asked that parts of that report be deleted?

Mr. McPhee. Do you want my personal opinion?

Mr. Rowland. Yes, I do.

Mr. McPhee. Well, obviously, they are embarrassing.

Mr. Rowland. Beg pardon?

Mr. McPhee. They were embarrassing.

Mr. Rowland. Would you elaborate on that?

Mr. McPhee. To Dow Chemical Co. Well, the multiple references to Dow as being the source of dioxin, the multiple appearances of Dow Chemical's name in connection with the dioxin issue.

Dr. Rowland. It was embarrassing to the company?

Mr. McPhee. This is purely personal opinion and speculation on my part.

Mr. Rowland. I read that a spokesman for Dow Chemical Co. was asked—was told, rather—that EPA asked that comments be given by Dow Chemical Co. Is that true? On this report?

Mr. McPhee. Nobody in the region to my knowledge asked Dow Chemical to make comments to the report .

Dr. Rowland. That statement is erroneous then, is that right?

Dr. Hernandez. Well, as I recall, they asked to see a copy of it and——

Dr. Rowland. They asked for it, then? They were not asked to make comments on it; is that right, Dr. Hernandez?

Dr. Hernandez. No. And I think the next—you know how people turn these things—I believe that they asked to have an opportunity to submit comments and I sent them out to the region. It's not our report. It's a regional report, and if they were the appropriate kinds of things that seemed reasonable to them, send your concerns out there. And I believe that happened. I don't know what the process was.

Dr. Rowland. Is that customary; that a company would ask to make comments on a report such as this?

Dr. Hernandez. Well, I think it was, you know, as I went through earlier, Dr. Rowland, this was my first experience with this sort of thing. The report was in the newspaper; it—the newspaper account said very clearly that Dow was reported to be the source of this pollution, dioxin, their plant area was saturated; and you can see the concern that that would raise in a company and their employees, who work there on the site and so on, and that they said, "Gosh, we want to see this report that you all have that allege this sort of thing," and—because my concern was could we substantiate it in terms of the information that we had. My quick reading of the report didn't seem to show that there was a proof that that was the case. Now, people back in the region may have had much more information to go on and so that the concerns of Dow being made to them about that particular statement that was in the press seemed to me a justified reason in sending this to them. I think if you put it in terms of what would they—let's say we had said, "No, you can't have the report." That would have raised many more questions in the employees of Dow and the State of Michigan's mind, who also asked for it, and the public in general about the thing. Making it available and seeing what the nature of that was, I think, provided an opportunity for a—not an inappropriate comment and concerns by Dow.

Mr. Rowland. There were some parts that were deleted?

Dr. Hernandez. Well, certainly as I understand it that there were and because there is a difference between those things, how much was deleted on the basis of Dow's request vis-a-vis what the region did with it after getting broader comments from their own people and from headquarters people, I don't know. And I think that's what Mr. Molinari has said, let's go through and track back on that.

Dr. ROWLAND. Well, if parts were deleted, would that make the company less culpable?

Dr. HERNANDEZ. You know, I'm not sure about culpable because there was no enforcement. This was a report dealing with dioxin from broad sources within that region. Now, if we had good proof that that plantsite was the source of dioxin that was coming into these fish, then we should never have changed our minds or deleted. I am not sure. I guess that was taken out; is that right?

Yes.

If we couldn't prove that allegation, then it was inappropriate to leave an unfounded, unscientifically based statement in there.

Dr. ROWLAND. Then you did not have scientific information; you did not have a good basis?

Dr. HERNANDEZ. I didn't know. All I said was, "Did we?" And, you know, that was my question.

Dr. ROWLAND. Well, Mr. McPhee, can you answer that?

Mr. MCPHEE. Sir, I am not a scientist.

Dr. ROWLAND. I understand.

Mr. MCPHEE. With respect to reviewing the technical validity of the compilation of the various reports that went into this report—and that's what this was, was a compilation of existing data—I couldn't begin to venture an opinion as to the validity of the underlying documentation.

Dr. ROWLAND. Mr. Chairman, I want to turn to something else for just a moment. The situation in Dallas that you referred to earlier about the lead poisoning.

Now, Dr. Hernandez, was there to your knowledge, any children found to be ill from lead poisoning that required treatment by doctors or hospitalized in this area?

Dr. HERNANDEZ. There were a few cases. Now, lead, as you know, has some manifestations besides just the higher blood lead levels, and—of lead toxicity, and I am not sure to what extent those were manifested in particular children. But there was a small group and I think it was about, oh, three or four or five children in that final survey.

Now, we went through two phases of this and I really don't remember the data on the first but I think the second one it was maybe on the order of six children who were above the 40 micrograms per decileter of blood, of lead in blood, and had some other indications, and they were placed on both a continued surveillance, as were all of them that exceeded the 30 level, and were placed perhaps on medication. I don't personally know about that. But they were put in the care of physicians.

Dr. ROWLAND. Was the illness of these children the thing that may have focused attention on a problem there; do you know?

Dr. HERNANDEZ. No. The attention initially came out of an EPA lead soils survey, and what they did out there in the region—and it was a good study. I want to say that to the credibility of the people who designed it and did it, that initial study was pretty well done.

It was not a well-stratified sample in the sense that you sample to find the plume. Now, we had to go back and do that in more detail. But it was a good study, by and large kind of a grid, and they went out from these plants and took samples and did a pretty good job.

It was those high lead soil samples that triggered concern. And——

Dr. ROWLAND. May I interrupt you there?

Dr. HERNANDEZ. Yes, sir.

Dr. ROWLAND. And ask was it after this point that the studies were done on the children and they were found to have this high lead level?

Dr. HERNANDEZ. Yes. And they were done in two phases, one, there was pretty much of a random sample. People were excited there. People were concerned in Dallas. And they collected a large number of blood lead samples from children and I think it was while a lot of them came from that area of concern, many of them came from all over town. As I recall, the number was something on the order of 75,000, which in a city like Dallas, is a lot of kids.

Then that study, because people did not come from a single area, was not well designed. We said, "Let's go do one that reflects the children down there near the smelters where the lead levels are higher," and that was the one that has just been completed and we have gone through a process with them.

Dr. ROWLAND. Am I correct in understanding that after it had been determined that some of these children did have high lead levels that the company agreed that there was a problem and they would do some cleanup, but you wanted to wait until some additional studies were being done?

Dr. HERNANDEZ. No. I don't think that's a fair characterization. I never was presented with a clear-cut cleanup option that said the companies will agree to do this, this and this and will remain liable for any future cleanup necessary and here's the agreement that we'll come to. There were references that one of the companies would do some of these things. Let me say one thing, because I am going to take the full responsibility for the decisionmaking on this as far as EPA is concerned, because while there were other governmental entities involved, as I have indicated—the city of Dallas, the State of Texas, CDC and certainly they—ours is an exposure concern; theirs is the blood level and the kids—I guess I believe that if we went out and started running bulldozers around, we would end up with a greater hysteria than we had already at the time that—without any clear-cut notion about what needed to be done.

Dr. ROWLAND. Am I to understand, then, that you were afraid that if you started cleaning up these areas or there was cleanup to take place, that it might create some hysteria?

Dr. HERNANDEZ. Well, if you went into it in anything other than a well programmed, well thought out, knew exactly what needed to be done—as I said, the original soil sampling was not totally definitive. We did some more work out there to determine where we feel the problems are, and if you had gone out and said, do it; let's take every place where there is a lead in soil level over—let's give it a number—500. We would have been out there tearing houses down, we would have been out there moving people out of houses; we would have been out there plowing up front yards and I just didn't feel that was an appropriate kind of end result.

Now, as I said, there was no clear-cut rationale or proposal ever made by one of the companies that I knew about that said, we'll do A, B and C, you know, we'll clean up that playground and we'll do this—a little bit of that was done. But all the things we'll do and then we're going to remain liable for everything else. There was never that kind of a thing.

I really believe, and still do, that the—because first you have to show that there is some kind of emergency; that you have some kind of a serious problem out there. The lead levels in soil were not sufficient. There is no target level out there that says you must act now because it is dangerous to go on. It doesn't exist that I know of.

I believe that a deliberate study mode where we took soil samples, samples in people's homes of dust levels, where —and blood levels that we did, would provide a good opportunity for people to make decisions within and to decide what kinds of actions were appropriate for the various kinds of alternatives that were open to us at that time.

Dr. **Rowland**. You do not believe then that the situation was such that additional people would become ill, additional children would become ill to any significant extent; is that correct?

Dr. **Hernandez**. Well, certainly that is always a concern. We issued advisories to people in terms of what kinds of things they should do in their lives to reduce exposure, and we really did those things, so I think we tried to minimize any kind of further exposure. There was enough in the press that—listen, if you said, did they hear about it—there were marches on city hall; a Congressman was fully involved in the sense of knowing the steps we were taking and why didn't they agree with us, but at least knew about them, and that's Congressman Frost——

Mr. **Levitas**. Would the gentleman yield?

Dr. **Rowland**. I yield.

Mr. **Levitas**. You are on a very good line, but I just wanted to put this into the record, and I have by the way discussed the matter with Congressman Frost and I did not get the sense from him that he was pleased with what was happening.

Dr. **Hernandez**. I don't say he endorsed it, but we kept him informed and he knew about the steps we took.

Mr. **Levitas**. I just state for the record he did not seem at all pleased with what was going on. But did you know in the summer of 1981 that the soil tests showed lead concentrations of a magnitude of 1,000 to 3,000 parts per million in the soil around these smelting plants.

Dr. **Hernandez**. Yes.

Mr. **Levitas**. You did know that?

Dr. **Hernandez**. Surely. That was our study.

Mr. **Levitas**. And do you know what is considered by medical evidence to be a level above which there is a threat to public health in terms of parts per million?

Dr. **Hernandez**. There are in the literature and the best one that I know of was a National Academy of Sciences study of about 2 years ago. I think 1980. But it may have been 1979, that discusses this issue of lead in soils and the discussion as I recall provides a kind of a level that says there may be problems above this level.

Mr. **Levitas**. And what is that level?

Dr. **Hernandez**. As I recall, it is on either the 500 to 700, some number like that.

Mr. **Levitas**. How about 300 parts per million?

Dr. **Hernandez**. It could be 300. I don't remember the 300. I would say 500 is my memory. But, you know, there's all kinds of numbers.

Mr. **Levitas**. Dr. Rowland was talking about the children. In one neighborhood, we understand that four children in this subdivision called Cadillac Edition, had been contaminated with lead and that one had been identified already as having suffered delayed mental development. Had that information been conveyed to you?

Dr. **Herhandez**. Are we talking about 1981?

Mr. **Levitas**. This is 1981.

Dr. **Hernandez**. No, I don't believe that I had heard of that specific case and certainly that's one of the reasons why that initial blood screening was done, to identify those problem children, but we did do that and I think that one happened by about October; I can't recall the exact timing, but it happened fairly soon, October, November.

Mr. **Levitas**. But it is your testimony that you were now aware of the information I've given about the employees, 110 employees—

Dr. **Hernandez**. No.

Mr. **Levitas** [continuing]. And the——

Dr. **Hernandez**. Sounds like I'm supposed to have seen that some place. Let me say——

Mr. **Levitas**. Well, no, I'm not playing games on this.

Dr. **Hernandez**. Let me say, I don't recall ever seeing it. I don't believe I ever saw it.

Mr. **Levitas**. Let me tell you why it concerns me. This was a press report. This was a press report that appeared in Dallas at the time, and knowing how Federal agencies work, including EPA, there is no doubt in my mind that somebody clipped that report and made it part of the file.

Dr. **Hernandez**. We have got dozens of clippings like that. So I am not sure that I read that one. I don't remember that piece.

Mr. **Levitas**. But the significance, as you see, it gets back to my concern and where you and I differ. If I had had the information of these high levels of contamination in the soil, plus the type of problems that were being manifested in these children in this area, in one community right next to these smelting plants, that would have been enough right there to say, hey, let's get in and do something; it's an eminent public health hazard; don't wait.

Dr. **Hernandez**. And I don't believe that an eminent, you know, that kind of emergency situation existed there.

We're virtually the same place today. We provided some guidance; we provided a detailed review of these—study of the children in the area; we've gone through and further characterized the soils; identified within homes sources of some of the problems and many of those things. But when you look at what we've recommended there, it is not characterized as a get-in and start bulldozing the land.

Mr. **Levitas**. Dr. Hernandez, again you have defined our difference in approach and attitude. I can tell you for a fact that if I had a child living in that neighborhood and I had the information as to the level of contamination in the soil and the health problems that other children in my neighborhood were suffering, I would have considered it an emergency situation.

Dr. **Hernandez**. And there are, you know, many public health people who were involved in looking at that decision from EPA, from CDC, who have a responsibility just like we do, and under Superfund a health responsibility on habitability. No one said, let's take emergency action to clean up all of that area. Now, the companies we are talking about doing something in playgrounds at a day school, they were not talking about moving people out of houses.

[Sound of buzzer.]

Mr. **Levitas**. Excuse me. It's a notice quorum call, notice for—would you call over to the—sorry——

Dr. **Hernandez**. You know, you characterized how you would feel about something. You know, we consulted with a lot of people about this, people who share that responsibility for making decisions on habitability. EPA does not make decisions on whether or not this area is habitable or not. Our decisionmaking has to do with monitoring, with statistic design of cleanup based on monitoring. Decisions on whether or not to move people out, to take them out of their houses is a public health decision, one that the Congress has said in Superfund belongs to HHS. And we agree to that sort of thing. Deciding on what is an appropriate cleanup level is part of our job, interpreting what CDC says. But in terms of cost effectiveness and remedial techniques and that sort of thing, there was no one who said, "take emergency action to move people out of those houses," to take such stringent actions that we would solve this problem.

A lot of those soil samples that you cite are not just in the playground; they are around those houses. What we did do was provide what we thought was adequate directions and cautions that the dangers would be markedly limited.

Now, let me give you a reason why I think some of those were effective. And remember, lead comes from a number of sources. It's not just out of soil, not just out of soil. It comes out of the air, out of the foods you eat, out of the dust in your house. So that there's a number of sources.

I believe that—and I have not looked at this statistically. I believe that the differences in the lead levels of some of those same children is probably lower in the second sampling than in the first sampling. I believe that some of those precautions and advice helped lower those things. I believe that over time, some of those things can be——

Mr. **Levitas**. They just vacated the quorum. You don't have to go.

Go ahead, Dr. Herhandez. And then—I've taken my colleague's time, and I will let him go on.

Dr. **Hernandez**. I believe that some of the problems associated with those homes there can be resolved. Some of them, as I recall—and I can't put a big hand it—some of the worst areas are in public housing areas. Those should probably be gradually moved out in at least the tier around there, grass planted, perhaps some topsoil moved away. They are looking at that kind of possibility.

People in their yards to plant grass, to put in vegetation so that dust off of the soils around there would not come through. I think that they will find that with some precautions and keeping homes clean, that the blood levels in those areas will probably not be significantly different from those in other people in the rest of Dallas. That doesn't say they are not going to be a little higher than some place else because they may be closer to freeways more congested and that kind of thing, more cars going up and down. But by and large, I believe that's a manageable thing. I think that it would have been irresponsible on my part to have said, this is an emergency situation; go out there and start moving people out of those houses. I think that would have been irresponsible on my part. We didn't have that kind of information.

Mr. LEVITAS. I think it was—well, I don't want to characterize it that way. I would simply say that if your concern is restoring credibility to the Environmental Protection Agency, there are a lot of people in America who would have liked to hear you say today on reflection, knowing what I know, I would have done everything I could as soon as possible to clean up what we could clean up, so that there would be this feeling that it is not going to get to the point where children are sick or more children are sick before something is done. I am simply addressing the question of perception.

Dr. HERNANDEZ. I agree with you. I would like to say that kind of thing, but I believe that the steps that we did take of cautioning people, of providing them with information, of putting into effect sampling programs, providing medical care for those where a level of concern was apparent. But that was the best thing to do in that case.

Mr. LEVITAS. And it is still your testimony that you did not know what negotiations were ongoing with the smelters concerning cleanup.

Dr. HERNANDEZ. Let me tell you again. There was no specific enforcement or negotiated settlement that was ever brought to me that said, "These people will agree to this in this explicit—sense of explicitness to do these things, to do these things, and that they will continue to be liable for anything else that needs to be done." There was no proposal that was brought to me by anyone that said, "Here's what we can do." And, you know, I think there could have been some other solutions.

Let me tell you another solution, another scenario.

We say, "Baloney, those lead levels look like that plume is caused by those guys, and I think there are some—now our sampling pretty well characterizes that—was caused by those people. Let's get out there and take immediate enforcement actions against them," which, you know, is a real possibility. And that was never in my—people didn't ask me about that one. Could have. Let me finish this——

Mr. LEVITAS. OK. I thought you were.

Dr. HERNANDEZ. No, no; I'm going to give you one more.

And we could have gone and done the cleanup, whatever it was. But I don't know where you would have stopped until you have gotten—if you said the trigger level is going to be 1,000, then we would have been out in people's houses, in people's yards, moving them out of homes—and believe me, the kinds of disruptions that you're—would come out of a contemplated action of people's lives could have been far more devastating than trying to follow a more rational approach down the line.

Mr. **Levitas**. You know you talk about upsetting people by taking the type of corrective actions you're talking about. I've already expressed my feeling. If it were my child living in that neighborhood, I would have thought it was an emergency, that something ought to be done. But the people out there thought it was a problem.

Didn't they have public protests and marches?

Dr. **Hernandez**. Absolutely; I said that.

Mr. **Levitas**. I know you did.

Dr. **Hernandez**. They were concerned.

Mr. **Levitas**. Doesn't that indicate that they were pretty highly activated and motivated and concerned and upset?

Dr. **Hernandez**. Correct.

Mr. **Levitas**. They even—I'm not suggesting there is any validity to this; they were suggesting that it was because it was a poor black ghetto area that they weren't getting the same type of attention they would have gotten if they lived someplace else. I don't believe there is any validity to that. What I am asking is, would a clinical physician say, well, we have got plenty of time; we don't have to be in a hurry to do the cleanup.

Dr. **Hernandez**. You know, if CDC had advised us to move in immediately to move people out, to reduce some kind of risk, I think we would have listened to them and taken that into account. I don't know of anybody who came out of CDC, and that's who we would look to in that kind of a recommendation, a habitability recommendation, said, "Let's move now to move people out of this area."

Dr. **Rowland**. And there were people from CDC——

Dr. **Hernandez**. Involved.

Dr. **Rowland**. Involved in this?

Dr. **Hernandez**. That's correct.

Dr. **Rowland**. And they made no suggestion that you move rapidly on it?

Dr. **Hernandez**. I think they made suggestions we move rapidly in terms of sampling, in terms of notifying people, but let's go back to in terms of some kind of emergency, remedial action, they did not.

Dr. **Rowland**. Thank you, Mr. Chairman.

Thank you, Dr. Hernandez.

Mr. **Levitas**. Thank you, Dr. Rowland.

Just a couple of other questions and I think we are about wound up for today.

Do you still have in your possession the files relating the Dallas lead matter?

Dr. **Hernandez**. We probably have a few.

Mr. **Levitas**. Do you have them in your files?

Dr. HERNANDEZ. We probably do.

Mr. LEVITAS. What other offices at EPA headquarters would have either files relating to this or copies of those files?

Dr. HERNANDEZ. I think the best source that would have everything is probably one that, a man named Walt Kovalich heads. The person who was head of that office and took the leadership responsibility was——

Mr. LEVITAS. Which office is he in?

What is his office title?

Dr. HERNANDEZ. I believe it is Toxics Integration, Office of Toxics Integration, in the Office of Toxic——

Mr. LEVITAS. Toxic——

Dr. HERNANDEZ [continuing]. Substances.

Mr. LEVITAS. Yes.

Dr. HERNANDEZ. You should have another name who you should talk to on this. It is a woman, Dr. Marilyn Bracken, who has recently left EPA, and she was the overall director of that process.

Mr. LEVITAS. We have already chatted with a couple of people about this.

But you have your files and I suggested that you look back through those to refresh your memory. I realize it's been a while.

Have you in the last—since you have been at EPA made a speech on the subject of "Scientific Bases for Regulatory Action," relating to what type of tests or studies should be relied upon?

Dr. HERNANDEZ. I have made many of those speeches.

Mr. LEVITAS. Did you make one at Harvard University in the last 2 years?

Dr. HERNANDEZ. Yes.

Mr. LEVITAS. When was that?

Dr. HERNANDEZ. Oh, probably—feels like October, November, December 1981, or maybe January 1982. Some——

Mr. LEVITAS. Do you have a copy of that?

Dr. HERHANDEZ. I didn't—I write all of my speeches on cards and never keep—we do have the cards, probably.

Mr. LEVITAS. Well, that might be helpful. I am interested in knowing what the thrust of your position was in that speech.

Dr. HERNANDEZ. In that speech, I think one very much that you heard me say two or three times today; that we need to make sure that in our decisionmaking on any chemical that we try to utilize all of the scientific body of information that is available to us in deciding how to regulate, how to limit its use, how to control its use, and so on; and that we not rely explicitly on one type of information, but that we try to involve everything we have. That doesn't mean you wait 100 years until you have more. But you take into account everything that you have at any one time, make your best cut, make some decisions on that.

Mr. LEVITAS. Did you take the position that you should wait until there are health-effects studies?

Dr. HERNANDEZ. Specifically—you mean health effects that you go out and count bodies, I guess is the——

Mr. LEVITAS. Yes.

Dr. HERNANDEZ. No; I think that by and large we have to rely on animal tests. We should interpret those tests wherever—when it sounds reasonable and there is many different kinds of scientific approaches to doing that. We should look at the various kinds of output that we have.

Mr. **Levitas**. In short, human effects studies, there are scientific basis for making regulatory decisions?

Dr. **Hernandez**. Yes.

Let me reference you to one that I think is an interesting one. We don't do it yet, but I think it is an interesting one.

When you look at animal tests, one of the things that in recent years we have been doing is trying to analyze the metabolic pathway and the various metabolic compounds that are formed in the process of putting this particular chemical through those test animals, and it is sort of like having a total tracking of the chemical to understand the mechanism that is involved.

There is the possibility that rather than looking at just the dose of chemical applied, that you might look at the—this particular chemical metabolism down here that ends up a metabolized form of the original chemical ends up—that has the adverse toxic health effect, and measure those, and use those in terms of dose-risk analysis because chemicals go by some kind of stokiometric relationship in some cases; some places it is transport across cell walls. If there is a repair mechanism and you are at some level, perhaps you can understand that mechanism of whether or not this material at some level may be a human carcinogen or may not be.

And those are the kinds of things I think we are moving to and a better understanding, and I hope that we can make use of all of that kind of information.

Mr. **Levitas**. If you would, I would appreciate your furnishing us copies of the notes if you still have them——

Dr. **Hernandez**. Sure do.

Mr. **Levitas** [continuing]. Of that speech made at Harvard, or any other published statements, or articles that you have written on this question of the testing, the evidentiary basis for regulatory action.

Dr. **Hernandez**. Yes, sir. Be very pleased to.

Mr. **Levitas**. Now——

Dr. **Hernandez**. Would you like to have one story out of that Harvard speech?

Mr. **Levitas**. Sure, I can take a story.

Dr. **Hernandez**. I was about to speak in the amphitheater up there——

Mr. **Levitas**. When was this, by the way?

Dr. **Hernandez**. This is October, November 1981 or January 1982.

And, of course, I had come from that school, but across the road or across the river, over on the other side at the Engineering School; and so, I knew some, a few of the people still there. In the amphitheater, there were 80 or 90 faculty members, graduate students, and the like. And I was just about ready to talk on risk assessment and this fellow comes up to me, and he says: "Hi, I'm Dr. Richard Wilson." And I said: "yeah, glad to know you; I've heard about you." And he said: "Yes, I thought you might like a copy of my book. It's on risk assessment."

Mr. **Levitas**. That's intimidating.

Dr. **Hernandez**. I'll tell you.

Mr. LEVITAS. I've had similar experiences and I am sure I found them no less enjoyable.

A couple of final questions to you, Dr. Hernandez.

On your calendars that we were examining, there are several references to meetings between you and one James Sanderson.

Dr. HERNANDEZ. Yes, sir.

Mr. LEVITAS. Did you ever meet with Mr. Sanderson subsequent to his leaving EPA in his official capacity?

Dr. HERNANDEZ. In a business sense? I'm trying to think whether I ever saw him socially.

Mr. LEVITAS. Well, leaving aside social visits.

Dr. HERNANDEZ. The answer to your question is no.

Mr. LEVITAS. So therefore your only meetings with Mr. Sanderson at EPA were in his official capacity other than if he came around for a Christmas party?

Dr. HERNANDEZ. No.

During the time that Mr. Sanderson was a part-time consultant to EPA, he was there initially as a part-time consultant, went back to his business and then came back to take a post at EPA, and so separating those things out.

Mr. LEVITAS. Right.

Dr. HERNANDEZ. During that time that he was acting as a part-time consultant to the Agency, and occasionally came—and I can't tell you the date; I'd have to look through it and I just don't remember—he came one day, and he said: "I want to take you out to lunch because I want to talk to you about a client that I have that has a potential EPA problem and I want you to know what it is." And he said: "I want you—I'm clearly putting on my business hat and want to do it outside the Agency so that we don't look like I'm Jim Sanderson." And he talked about a Denver Water Board issue that to my knowledge has never become an EPA regulatory or action issue in any way.

Mr. LEVITAS. Did you ever discuss any of the following with Mr. Sanderson: the Seymour Recycling case in Indiana, or Chemdyne?

Dr. HERNANDEZ. No, sir.

Mr. LEVITAS. Chemdyne?

Dr. HERNANDEZ. No, sir.

Mr. LEVITAS. Love Canal?

Dr. HERNANDEZ. No, sir.

Mr. LEVITAS. Any type of permitting decisions?

Dr. HERNANDEZ. No.

Mr. LEVITAS. In——

Dr. HERNANDEZ. In solid waste and stuff like that.

Mr. LEVITAS. Yes, incineration permits.

Dr. HERNANDEZ. No.

Mr. LEVITAS. Mr. Brown, you testified before our committee, full committee, back in February, I guess about the 15th——

Mr. BROWN. I'll take your word for the date, sir.

Mr. LEVITAS. OK.

Mr. BROWN. I don't think it was in February. I don't think—it could have been; yes, sir.

Mr. LEVITAS. We were talking about shredders at that time.

Mr. BROWN. Yes, sir. I remember that one.

Mr. **Levitas**. The specific date is not important. I was just using that as a frame of reference. The reason I am asking you this question, so you will understand I am not—the question has arisen.

Mr. Albosta asked a number of questions about whether EPA consultants had access to files that were being denied to the committee.

Do you recall that line of questioning?

Mr. **Brown**. No; but, you put the question wherever you would want——

Mr. **Levitas**. The question that I am getting to relates to this. There were a series of questions that Mr. Albosta was asking of all the witnesses, did you know whether other people were getting access to files that were being denied to this committee. And one of the line of questions was, did you know of any consultants who had access to enforcement files that were not being turned over to this committee. And everybody, so far as I can recall, said no.

The letter—the memorandum from Mr. Currant to yourself concerning Dr. Pollata made reference to—Dr. Pollata, you recall was a consultant for a while to EPA——

Mr. **Brown**. That's correct.

Mr. **Levitas** [continuing]. Made reference to Dr. Pollata's aggressive involvement in influencing certain decisions with respect to dioxine levels and some other matters.

Mr. **Brown**. That's correct.

Mr. **Levitas**. Now, the question—I'm just trying to clear it up for the record.

Having that called to your attention, would Dr. Pollata's access to information and participation in those discussions as memorialized by Mr. Currant in his memorandum to you have been access to information that was being denied to this committee?

Mr. **Brown**. I don't know. We would have to check with Mr. Currant or his staff members. I have never been in on a meeting with Dr. Pollata on any enforcement matters.

Mr. **Levitas**. All right.

Mr. **Brown**. Now, maybe Mr. Currant's staff members could say if there was some—what Ed Currant brought to my attention was he felt as reflected in this memo that Dr. Pollata's view of acceptable levels and all were not in keeping with agency practice. Now, I don't know from anything Ed told me that that would indicate that he had access to enforcement files or anything like that, to make those views known.

Mr. **Levitas**. If you would go back and take another look at that memorandum, I am sure it is not——

Mr. **Brown**. Oh, I am sure—I would say that—well, Ed, of course, was the head of the division. We would have to find out the people who had talked to Ed that would have had those complaints; or possibly Ed had been in on the meetings himself; I don't know.

Mr. **Levitas**. My only request is that you go back and take another look at that memorandum and ascertain whether or not that would have revealed or suggested Dr. Pollata's having access to those types of files that we were talking about.

Would you do that for us?

Mr. **Brown**. Surely.

[The following was received for the record:]

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, DC 20460

REC'D MAR 2 5 1983
Subcomm[illegible]
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Committee o[illegible] Works
and Transportation

MAR 2 5 1983

OFFICE OF
ENFORCEMENT COUNSEL

Honorable Elliot Levitas
Chairman
Subcommittee on Investigations
and Oversight
Committee on Public Works
and Transportation
House of Representatives
Washington, D.C. 20515

Dear Chairman Levitas:

At the hearing before your Subcommittee on Wednesday, March 16, 1983, you referred to a question by Congressman Albosta at a February 16 hearing before the full Committee. The thrust of your inquiry was whether Dr. Arthur Palotta had access to enforcement sensitive documents being withheld from your Subcommittee. When I indicated I could not remember the question from the previous hearing, you asked me to review my answer in view of the memorandum from Edward Kurent, then Acting Associate Enforcement Counsel, to me, subject: Potential Problems in Process and Potential Outcome of Determination of Cleanup Levels for Dioxin in Soil, dated September 17, 1982. You were kind enough to allow me to return to my office to review the memorandum and submit a further answer for the record.

I reviewed the transcript of the prior hearing and the only reference to access by consultants to enforcement sensitive documents that I could find involving me was on pages 77 and 78 where I told Congressman Roe that consultants in fact created enforcement sensitive documents in some instances (lines 1765 - 1771). Accordingly, I do not wish to add anything to that answer.

I spoke to Mr. Robert Prolman of your staff and we agreed that I would submit for the record any information that I could discover about Dr. Palotta's access to enforcement sensitive material. I have again read the memorandum from Mr. Kurent. Nothing in the memorandum leads me to conclude that Dr. Arthur Palotta saw documents that were withheld from your Subcommittee because they were enforcement sensitive. However, I asked my Office of Enforcement Counsel - Waste and the heads of the offices mentioned in Mr. Kurent's memorandum to me as having concerns with Dr.

Palotta's actions (Offices of Emergency and Remedial Response (Superfund) and Waste Programs Enforcement) to determine if any documents to which Dr. Palotta had access had, in fact, been withheld from your Subcommittee.

I am informed by my Office of Enforcement Counsel - Waste that the only document to which Dr. Palotta had access which might have been considered enforcement sensitive at the time was the September 17, 1982, memorandum from Edward Kurent to Conrad Kleveno, subject: Enforcement Concerns In the Process and Outcome of Determinations of Cleanup Levels for Dioxin In Soil. This document was made available to the Congress in November 1982.

Mr. Hedeman informed me that he was aware of no enforcement sensitive material in the Office of Emergency Response and Remedial Action to which Dr. Palotta had access. Mr. Lucero stated that Dr. Palotta had access to no enforcement sensitive material in the Office of Waste Programs Enforcement in connection with the Missouri dioxin sites. However, Dr. Palotta did work on the health effects assessment concerning asbestos at a site at Globe, Arizona and, in the course of that work, did have access to the case development plan, a document preliminarily determined by Mr. Lucero to be enforcement sensitive. Also, Dr. Palotta had access to enforcement sensitive material in his review of data and attendance of meetings concerning the DDT problems near Triana, Alabama.

In my capacity as Acting Assistant Administrator for Solid Waste and Emergency Response I requested that the documents in Dr. Palotta's office be inventoried after the decision was made to terminate his consultant status. I am informed that, aside from the Kurent memorandum discussed above, no enforcement sensitive documents were found in that review.

To the best of my knowledge, based upon the inquiry I have outlined above, these are the only instances in which Dr. Palotta may have had access to enforcement sensitive material which may have been withheld from your Subcommittee in the past.

Please let me know if there is anything else we can do to help complete your record in this matter.

Sincerely,

Michael A. Brown
Enforcement Counsel

Mr. LEVITAS. And if there is anything you want to supplement, based upon that review, please submit it to the committee.

I think we're almost over, Dr. Hernandez. I want to get into one question--series of questions to you and Mr. Cannon dealing with resources.

There has been considerable testimony about the level of resources for the funding and staffing of Superfund. And the Agency's budget proposals for the past few years have made drastic reductions in funding levels in most programs and significant reductions in staffing as well. And if I understand it correctly, the current budget proposals will result in a reduction of about 30 to 40 percent, as compared to 3 or 4 years ago.

When Mrs. Gorsuch testified before the Appropriations Subcommittee last year, she said that "* * * the budget represents more than efficiency, more than Federal scrutiny, it is a rededication of resources away from government for the sake of government and towards environmental results."

She went on to say: "* * * we intend to concentrate on our statutory mandates, reduce backlogs, implement the original intent of Congress by delegating to the States wherever possible an improve and enhance the science behind our programs. This I believe is a good budget and an effective budget and I am proud of it."

Again, in her testimony this year, before she departed, there were a number of cuts and there was again a reassertion of the amount of Federal support to State agencies to carry out the types of things; they were being cut as well even though they were being delegated these responsibilities.

You testified before our Water Resources Subcommittee last year and you said, I think we can do a good job in 1983 with what we have asked for; we will reassess our needs for 1984 as they come along.

Now, we have had testimony, and the documents we have examined in executive session have revealed that there have not been enough resources to do the job. We have seen memoranda from people like Mr. Currant who recently left the Superfund Program. And he observed that the lack of staff has reached crisis proportions; and our own subcommittee investigators have noted the same thing.

Those enforcement-sensitive documents have made some very startling revelations about the impact of lack of resources.

Now, my question is, Dr. Hernandez, are you satisfied with EPA's current level of resources, and are you confident that you have what you need to carry out the environmental mandates that the Agency has?

Dr. HERNANDEZ. Mr. Chairman, there are some problems in program areas that need additional resources both in 1983 and into 1984. I've looked at this in a way and said, "how far were we off in terms of hitting the right number in what we need?" I believe that these are on the order of 1 percent or 2 percent of that operating budget that we really run the agency on State grants aside, and the Federal grants to construction projects aside, just that operating fund.

We are off somewhere; it's such a tight budget that when you're off a little bit, you do notice it, so we do have some needs. And we have prepared a request and will transmit it—or have transmitted a piece of it to OMB for their consideration; and we are revising or will review that.

Mr. LEVITAS. Am I correct that your 1984 budget justification papers indicate that you are requesting only about 5 additional work years for the Superfund Program?

Is that correct, Mr. Cannon?

Mr. CANNON. I think that's——

Mr. LEVITAS. Well, does that appear to be responsive to the crisis conditions of the sort that Mr. Currant has described?

Dr. HERNANDEZ. This was our original 1984 request; is that correct?

Mr. LEVITAS. That's right.

Dr. HERNANDEZ. Let me say that that particular one is under review; we have made a recommendation or will be further processing a recommendation for some additional people in the enforcement area, and——

Mr. LEVITAS. It just seems to me we have been talking about the problems and the need and the delays and the backlogs——

Dr. HERNANDEZ. I know. And we have some problems, and I said we need to increase the budget some.

Mr. LEVITAS. It is a minimum response, it seems to me, to talk about 5 additional work years for a program as important as Superfund.

Mr. Cannon, could you help us on this a little bit?

Mr. CANNON. One thing that needs to be pointed out, I think the concerns of Mr. Currant's memorandum had to do with resources with respect to the Office of General Counsel and the enforcement programs and not directly related to Superfund.

With respect to Superfund, I think it is not as people intensive as it is dollar intensive, and our own view is that we have adequately funded the Superfund activities. There are a couple of things that are going on at the Agency that may help us with this. First of all, we are undergoing our regular midterm review of the—that we do about halfway through the fiscal year to see what our operating needs are, and the second thing that we are doing is an extensive management review of the entire Superfund and Hazardous Waste Program, which will be completed in, as Dr. Hernandez spoke about earlier, a few weeks from now, to which we expect to address these questions, particularly the resource questions.

Mr. LEVITAS. In the set of memoranda concerning the Administrator's accountability system, at the end of the third quarter of fiscal year 1982 and at the end of the fourth quarter of fiscal year 1982, there is a—there appears as a significant slippage or slowdown in the program, especially between the obligation of Superfund moneys and the expenditure, and that appears to our committee that has been looking at the Clean Water Act Construction Grant Program for years to be the same type of phenomenon we've seen there, where because of limited staff and a myriad of other resource problems, although not all resource problems, it has appeared that many projects are poorly prepared and rushed through at the end of an obligation so as to submit the funds, and then after they are obligated, the Agency staff and others stall the project while they straighten out the bugs.

Are we seeing a duplication of this now in Superfund?

Mr. CANNON. We hope you don't see a total duplication of that, of the experience that we had with construction grants. Mr. Hedeman might more accurately address that, but it is true this is a problem that surfaced in our—through our—at least to us in the Office of Policy and Resource Management through the accountability system.

Mr. HEDEMAN. One of the realities we are having in the program is that we obligated money for a number of remedial investigations and feasibility studies for sites toward the latter end of fiscal year 1982. And they generally take about a year to accomplish, and so they are so to speak in the pipeline and the remedies that come from those studies will not really be known until the summer months.

Those remedies equate to the higher cost of construction, and so we will be obligating in this program in fiscal year 1983 a majority of the money for construction in the last several months of the fiscal year only because we were not able to get the projects into the pipeline in terms of doing the studies to select the alternative until the latter part of the last fiscal year. I realize that's complicated. It's a matter of——

Mr. LEVITAS. No, I understand what you are saying. Again, that is a more professional description of the problem that I think I have described to Mr. Cannon. I think it is a more technical description of what I am perceiving as the problem, and it is a problem that begins to feed on itself after a while, it seems to me.

Did—oh, you were talking about Mr. Currant's shop, which was litigation. Wasn't he also handling litigation for Superfund?

Mr. HEDEMAN. Yes, sir.

Mr. LEVITAS. The one last question I have got.

At one point, I believe I read or heard that Mrs. Gorsuch, which she then was, expressed the belief that the Superfund would not have to be continued beyond 1985. Dr. Hernandez, do either you or Mr. Cannon share that view?

Dr. HERNANDEZ. I think that the best thing that we can do is provide the Congress with our best estimate in that report that is required. What will remain out there, what kinds of exposures, what kinds of materials, based on these assessments that we are doing, and the society, you need to make that kind of a decision, on whether or not it would be continued.

Mr. LEVITAS. Well, you've got an opinion on that right now, don't you?

Dr. HERNANDEZ. Well, in all candor, it is a little early. I think we need to see, you know, if you say, what are we going to get done, and it is clear that through the process we are going to get done something like a hundred ourselves, by the enforcement process we will get a few more, by the negotiated process. Let's put that total package at 300, maybe, generously—300——

Mr. LEVITAS. Well, what does that drive you to?

Dr. HERNANDEZ. Then I say, let's look at what we have as we go down there and then let's make some societal decisions on what it is, what we need, what kind of format.

Mr. **Levitas**. Just on the basis of what you said right now, surely you've got an opinion as to whether we are going to need to continue the Superfund Program and the fund itself.

I've got an opinion. I think——

Dr. **Hernandez**. I think what—you would say, do you have a feeling of what is going to happen.

Mr. **Levitas**. No. I want to know do you have an opinion——

Dr. **Hernandez**. Because there is a difference. There's a difference in——

Mr. **Levitas**. It would be hard for me to believe that anybody who has listened to any of this testimony doesn't have an opinion on that question; in fact, I would think that may be one of the few things we are completely in accord on.

Dr. **Hernandez**. Yes.

Let me say, I believe that there will need to be some kind of either combination of State and Federal source for some time out in the future.

Mr. **Levitas**. Again, I am trying to be constructive——

Dr. **Hernandez**. Yes.

Mr. **Levitas**. You are not giving a lot of assurance or reassurance to the people in America by saying what you just said to me.

Dr. **Hernandez**. Gosh, that was a pretty positive one.

Mr. **Levitas**. You want to make the people in America feel a little bit more comfortable. Just tell them flat out that you know that we are going to have to extend the Superfund Program and get on and continue the work of cleaning up the hazardous waste sites. That is what they are concerned about.

Dr. **Hernandez**. I think that we need to continue to work on those sites, whether or not it is under this mechanism or some other one.

Mr. **Levitas**. Mr. Cannon, do you have any feelings on this subject?

Mr. **Cannon**. There is no question, from our studies we know there is a continuing problem and there is going to have to be some Federal response to it. I would hate to commit to the identical piece of Superfund legislation because I would hope that throughout this process of our studies that we are going to provide you, not just our management studies, but the study that is due from us to you that we have come to possibly even a more constructive result than what we have now.

Mr. **Levitas**. That's an encouraging and positive response.

I want you to know—I don't know how closely you have been following our hearings, but a couple of weeks ago we had difficulty getting some of the EPA witnesses to suggest to us that there is room for improvement in the program, and I am glad to hear that we are able to talk about this candidly.

Mr. **Cannon**. Well, let me note for the record that while we know we have problems, we are not saying that the statute is perfect, and we are hoping that we can through this mutual effort here come to really a better resolution.

Mr. **Levitas**. There is no question, as I said in my remarks at the beginning of today's hearing, and on every occasion, that the statute and the implementation of the statute have got to be looked at carefully. That's what we are doing. At the same time, we are chasing these jackrabbits as they keep jumping up and down the path.

The gentleman from New York.

Mr. **Molinari.** I listened to the exchange rather carefully, Dr. Hernandez, and, frankly, I got the impression that you really have reservations about the continuation of Superfund.

Dr. **Hernandez.** I think per se I think we ought to examine it, see what we have done with it. Is there a better mechanism? Is there one that will take care of problems more quickly; and I think there is a lot of questions that we could answer when we are a little further down the road in that sense.

Mr. **Molinari.** Have you ever taken a position on or given consideration to, discontinuing the Superfund, as we know it?

Dr. **Hernandez.** No.

Mr. **Molinari.** Well, why do you have so much difficulty in answering his question as to where we go after the existing legislation expires?

Dr. **Hernandez.** Let me be very candid with you.

What I don't want to do is speak for this administration in terms of future policy with respect to that particular program.

Mr. **Molinari.** I don't think he asked that. I think he was only asking for your own opinion.

Dr. **Hernandez.** Well—but I was being careful not to do that, because, first, I don't have that kind of sense of carte blanche in terms of the administration's position. Let me try it one more time and see if we can't come up with it.

There will clearly be needs when all that Superfund money is utilized, whether it takes to 1988 to do it or not, but there clearly will be needs that will continue to exist. The order of magnitude, how to go about that in terms of that State-Federal participation, we should address, and what that program should look like in the future needs to be that kind of——

Mr. **Molinari.** Well, Dr. Hernandez, have you advocated turning over a greater role to the States to carry the program out?

Dr. **Hernandez.** To do the cleanup work?

Mr. **Molinari.** Well, yes, sir.

Dr. **Hernandez.** Well, the answer is yes, because I believe they do a better job in many respects; they are right close to the problem there, and 60 percent of our projects are being run by the States right now.

If you mean a greater sense of responsibility in terms of paying for it, clearly there is a lot of reluctance and it would be very disuniform across the Nation unless we had the kind of thing we have under Superfund right now, some kind of national fund.

Mr. **Molinari.** How would you rate New York State in terms of effectiveness in dealing with this type of problem?

Dr. **Hernandez.** I just really don't know. I would have to have—I don't have any personal relationships there at all.

Mr. **Hedeman.** It's generally very good. They have within the last year moved to implement their own Superfund. I was advised last week they are having funding problems with that fund. It is not taking in the money as rapidly as they thought, but——

Mr. **Molinari.** I have been somewhat heavily involved in a couple of sites in which New York State has done a horrible job.

I've heard some officials from EPA state that New York State and New Jersey are two of the best in terms of dealing with the issue. Well, if they're the best, we really have problems; and if the administration or you, Dr. Hernandez, is giving serious consideration to divesting yourself of additional responsibility and to turning it over to the States, I think you are going to have a problem with this Member.

Dr. **Hernandez**. Nobody is suggesting that at all. I would—having the States run these projects develops an expertise in their offices and gives them some more wherewithal not only to deal with that issue but future problems; so I really believe in building up that kind of manpower capability there. Nobody is going to suggest that the Federal role today should change under Superfund as we now have it under this fund.

Mr. **Molinari**. Then you do advocate the continuation of Superfund?

Dr. **Hernandez**. For the time being, within the statutory framework we now have.

Mr. **Molinari**. No, I am talking about on the 1985 expiration date.

Dr. **Hernandez**. We will come back here with recommendations. You know, clearly there is going to be a need out there. What that form should take in the future, let's deal with it then.

Mr. **Molinari**. I have no further questions, Mr. Chairman.

Mr. **Levitas**. Thank you, Mr. Molinari.

I have no further questions at this time.

I want to thank you all for being with us today. Dr. Hernandez, I recognize this is your first appearance as Acting Administrator of EPA; I hazard a guess it will not be your last. My sense is that the feeling of Congress is that we are most anxious to get EPA back on track so it can do the job that all of us in this country, on a bipartisan basis, want to see it do; and anything that you can do and any contribution you can make, not only to deal on a day-to-day basis with the problems that you have and the responsibilities you have as Acting Administrator, but to get the attitude, the philosophy and concerns of this Agency highly directed and motivated on public opinion and it just needs to be addressed desperately.

Dr. **Hernandez**. Mr. Chairman, running EPA is probably one of the toughest Federal agencies. If there is another one where we are more into people's lives and into their health and into their well being and that personal fear that they have, I don't know of one.

It is a very difficult process we go through because it is in that fishbowl of life. Decisions are always easy to dispute, easy to say, I'd have done something different.

I came out of a public health organization. You should have no fears. That's my own foundation, my own historical background, of being someone who has spent about 7 years in a health department organization, fundamentally trained to protecting people's lives and health; and we will still have some of the best people amongst us here to carry out that work. We are the Environmental Protection Agency and I think the American people can count on us.

Mr. **Levitas**. We are going to pursue our investigation thoroughly, objectively, to the end, so we can get to the bottom of the Superfund question and the role of the Environmental Protection

Agency; and as I said earlier, I just wish we could put back into our dictionary words like, we made a mistake; we set out in the wrong direction; now is the time to change. I assure you, if that were done, the support of this Congress and the American people would be forthcoming. If it doesn't happen, then changing personnel isn't going to make a tinker's damn of difference.

So anyway, I thank you very much, and your colleagues and associates; you have been extremely helpful and you have advanced our investigation, which we hope will result in appropriate recommendations to improve the cleanup of the hazardous waste sites in this country.

There being no further business, the subcommittee stands adjourned, subject to the call of the Chair.

[Whereupon, at 3:40 p.m., the subcommittee recessed, to reconvene subject to the call of the Chair.]

[The following additional items were received for the record:]

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

May 29, 1981

OFFICE OF
PESTICIDES AND TOXIC SUBSTANCES

MEMORANDUM

SUBJECT: Dr. Milt Clark's Dioxin Paper

FROM: Paul H. Brown PH Brown
Co-Chairman
Chlorinted Dioxins Work Group (TS-777)

TO: Karl Bremer
Toxic Substances Coordinator
EPA Region V

The Chlorinated Dioxins Work Group has concluded its review of the draft dioxins paper by Dr. J. Milton Clark. In most cases, members of the group wrote their comments on copies of the paper; I have combined these comments onto one copy. Two of our members commented in separate memoranda, which I have attached.

I have one additional suggestion: separate the scientific (pp. 1-27) from the regulatory policy (pp. 27-28) sections of the paper and include the regulatory policy issues as a separate cover memorandum from you describing the paper. This would help keep the policy materials safer from FOIA release and emphasize the responsibilities of agency decision-makers -- who, presumably, are more reluctant to alter the scientific portions than the regulatory issues portions.

Overall, the paper is an excellent summary of the available environmental monitoring data. Please keep me involved in Region V dioxins work, if at all possible.

Attachments

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF
PESTICIDES AND TOXIC SUBSTANCES

MEMORANDUM

Subject: Dioxin Report

From: Larry E. Longanecker
Chemical Control Division (TS-794)

Thru: Paul Brown, Chairman
Chlorinated Dioxin Work Group

To: Karl E. Bremer, Toxic Substances Coordinator
Region V

I have several major reservations about this report, especially the conclusions drawn.

First, the report concludes with a sweeping statement that the acceptable daily intake of TCDD should be 10^{-5} ug/kg/day. The citation for this statement is an EPA letter. Beyond the fact that this letter is not available for inspection by the public (or me, for that matter), the study upon which the calculation is purportedly based does not even appear in the list of references. Actually, there are at most 5 references to scientific literature on the health effects of TCDD, none of which could support findings of the magnitude so readily stated in this report.

This leads to my second objection. There is in this report an overabundance of self-reference. I don't believe that EPA is well-served by quoting itself on all the major questions of dioxin. More primary literature on health effects is needed if this report is to substantiate health hazards.

Third, the report may cause overreaction. Uninformed as I am, and after reading page after page about the ubiquitous presence of TCDD (most of the scientific literature cited deals only with the latest measurement of TCDD in incinerators, etc.), I would have to conclude that I should eat no fish caught in the U.S. (and perhaps avoid all incinerators). Is this really the impression we wish to make with the public? If it is, I suggest it is one best left to the rulemaking proceedings of EPA (or FDA), where critical external review of important health findings can be guaranteed.

Finally, it is important, when reviewing a report, to know the purpose to which the report is to be put. This report is titled a "Review of . . . Sources and Effects," a title not reflecting the very strong conclusory statements about prohibiting the eating of fish caught in several Michigan rivers and, by implication, the Great Lakes as well.

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF SOLID WASTE

DATE

SUBJECT Comments on Dr. Clark's report on PCDDs and PCDFs.

FROM Judith S. Bellin, Ph.D
Waste Characterization Branch
Hazardous and Industrial Waste Division (WH-565)

TO Paul Brown
TS777

p5&6 o Photolysis - The degradation of dioxins by photo reduction is not adequately discussed, especially the field experience (Sytec?) which we do have. I would like to see a reference for the statement that photoreduction is appreciably slowed by adsorption. There should be a reference to Ayers' work on ruthenium tetroxide oxidative degradation.

p8 o The chronic toxicity of PCDFs: there should be a discussion of McNulty's work (Food Cosm. Tox. 19: 57-65: 1981.

p10 o Discussion of work in Australia should include the recent science article (April 17, 1981) showing increased frequency of club foot and qenitourinary malformations.

p27 o RCRA disposal requirements are not adequately explained. The TSCA 6d rule is not mentioned (I have appened appended some material which may be helpful to Dr. Clark).

o There is no reference in the report to the Vertac experience - this may not be relevant to this report (I dont know whether the author intends to confine the story to the Great Lakes area.).

o The risk assessment discussion should ~~di~~ be separated from that of RCRA. The ADI for TCDD (WQC) is 10^{-5}, not 10^{-4} and was based on reproductive effects.

o Reference 35 is not a peer-reviewed Journal. It is not adequate to support the extensive quote on Dow's conclusions. The last sentence of this paragraph is meaningless.

p28 o The WQC is (was?) $2.1x10^{-10}$. (ref 14, as quoted, is outdated).

Reference Changes:

Ref.12 - part is missing
Ref.14 - should now read: "Ambient water Quality Criteria for 2,3,7,8........EPA 440/5-80-072, February 1981"

IV. Present regulation of TCDD wastes.

A. RCRA
   1. §261.24: D016- 24D   D017- Silvex
   2. 261.33f: lists chlorophenols, 2,4D and 245T esters and salts.
   3. 261.32:

K042: residues from distillation of tetrachlorobenzene in the production of 245T (note the restriction!- some 245TCP is presumabl used for other purposes). Toxicants: o-dichloro and hexachlorobenzene

K043: 2,6DCP waste from the production of 24D (note the restriction- many other pesticides are mfrd from DCP -see Table I). Listed toxicants: 24DCP, 26DCP, 246TCP.

K099: untreated wastewater from the production of 2,4D (see note under K042). Listed Toxicants: 124DCP, 246TCP.

B. TSCA dd rule.

1. covers wastes from the production of 245TCP and/or its pesticide derivatives, or wastes from the production of other compounds on equipment that was at some time used to produce 245TCP and/or its pesticide derivatives.

2. disposers of such "new" (post May 12 1980) wastes must test the weaste (I have not been able to find any data except for Systec), and, if they find "detectable" TCDD must notify EPA 60 d prior to intended disposal. Notification is to include information on intended method os disposal which may include land disposal.

3. The regulation excludes persons disposing of wastes containing TCDD at facilities permitted for disposal in a RCRA permitted facility. Since there are none of those as yet, if TSCA evaluators do not approve of the disposal method, these wastes must remain on-site, a problem. Also, when permitted facilities are available the effect would be to permit land disposal which (see below) is not desirable.

[illegible]

5. Some materials, e.g. soot from transformer fire, are not covered, and, as of now could be dumped anywhere.

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION V

DATE: 1 MAY 1981

SUBJECT: Dioxin Report

FROM: Karl E. Bremer Karl E. Bremer
Toxic Substances Coordinator

TO: Members of Dow Task Force

Please review the final draft of the Dioxin Report by Milt Clark for technical accuracy and offer comments no later than May 15, 1981. As this report will be released to the public it is extremely important that it be carefully reviewed. Thank you for your assistance.

cc: D. Kee/AHMD
D. Barnes
G. Amendola/SAD
V. Saulys/GLPO
B. Maddox/Water
C. Ross/SAD
T. McPhee/EWPE
T. Barney/EP
M. Mutman/EWPE
S. Gardebring/E

Please use replacement language in your comments. Thank you.

I have reviewed this document as to its accuracy. I have made some obvious necessary corrections and against your wishes, unfortunately had to editorialize in some places.

P Edes Rosins
5/8/81

EPA FORM 1320-6 (REV 3-76)

A Review of Polychlorinated dibenzo-p-dioxins (PCDDs) and Polychlorinated dibenzofurans (PCDFs): Sources and Effects

I. Introduction

In the last several years, concern over the environmental and public health effects from the use of 2,4,5-Trichlorophenoxyacetic acid (2,4,5-T), a herbicide, has increased. Chemical products with 2,4,5-T were shown to contain a trace contaminant 2,3,7,8-Tetrachloro-dibenzo-p-dioxin (TCDD). This chemical, commonly known as dioxin, is possibly the most toxic chemical synthesized by man. Many commercial products contain chlorinated dioxins, technically known as polychlorinated dibenzo-p-dioxins (PCDDs) and related compounds, polychlorinated dibenzofurans (PCDFs). Evidence exists that PCDDs and PCDFs may also be released to the environment by combustion processes. This report (1) summarizes the sources of PCDDs and PCDFs; (2) lists the physical, chemical, and biological properties of these compounds; and (3) discusses those studies involving PCDDs and PCDFs in the Great Lakes area.

II. Sources of Polychlorinated dibenzo-p-dioxins (PCDDs) and dibenzofurans (PCDFs) in Commercial Products

A. 2,4,5-T (phenoxy acids)

The dioxin problem was first observed in connection with the teratogenic effects observed in laboratory animals from 30 ppm of TCDD in the herbicide 2,4,5-T (1). TCDD is formed in the manufacture of 2,4,5-TCP ~~2,4,5-T~~ from 1,2,4,5-tetrachlorobenzene, especially if production temperatures exceed 160°C(2). Drums of Agent Orange stored in the USA (Gulfport, MS) and in the Pacific (Johnston Island) prior to 1970 were ~~have been~~ reported to have TCDD levels ranging from 0.1 to 47 ppm (3). Only trace levels of other PCDDs and PCDFs were found. Currently, producers of 2,4,5-T claim that their products contain less than 0.1 ppm of TCDD (2). On February 28, 1979, the use of 2,4,5-T and Silvex (2,4,5-TP) containing products was immediately suspended

[an intermediate to 2,4,5-T production

NO, IT ISN'T

There are currently no producers of 2,4,5-TCP, hence, no 2,4,5-T is produced due to findings of increased spontaneous abortions in Alsea, Oregon, an area where 2,4,5-T had been extensively used in forestry to control undesirable plant growth. (4,5).

B. Chlorinated Phenols

trichlorophenols, tetrachlorophenols,

there is only one PCP.

PCDDs and PCDFs have been found in ~~tri, tetra,~~ and pentachlorophenol~~s~~. The quantities of these compounds and their isomeric forms depend upon the reaction process and conditions. Current concentrations of 2,3,7,8-TCDD in 2,4,5-trichlorophenol (2,4,5-TCP) and its sodium salt are less than 0.1 ppm (6). Analyses of PCDDs in commercial grade pentachlorophenol~~s~~ (PCP) produced by by various manufacturers during the years 1975 to 1977 found hexa-CDDs (HxCDDs) [non-detectable(N.D.) to 520 ppm], hepta-CDDs (HpCDDs) (125 to 180 ppm) and octa-CDDs (OCDDs) (N.D. to 3600 ppm) (7). No TCDD was found in commercial PCP. PCDFs were also detected including hexa-CDFs (HxCDFs) (9 to 90 ppm), HpCDFs (70 to 400 ppm) and OCDDs (55 to 260 ppm). There are several isomeric forms of HxCDD, the major isomers being the 1,2,3,6,8,9 (40-50%); 1,2,3,6,7,8 (20-40%); and 1,2,4,6,7,9 (20-40%). The isomeric forms for HpCDD are 1,2,3,4,6,7,9 (60%) and 1,2,3,4,6,7,8 (40%). Commercial PCP also contains 5-10% 2,3,4,6- tetrachlorophenol ≤0.1% 2,4,6-trichlorophenol and about ~~8%~~ 6 of various chlorinated phenoxy phenols.

Unline

III. Sources of PCDDs and PCDFs from Pyrolysis, Combustion and Photolysis

A. Formation of PCDDs by Pyrolysis of Chlorinated Phenols

1. The burning of materials impregnated with commercial 2,3,4,6 tetrachlorophenates yielded a total of 150-1000µg of PCDDs/kg of chlorophenate. The PCDD isomers detected ranged from various TCDD~~s~~ to HpCDDs. The 2,3,7,8 TCDD and 1,2,3,7,8 penta-CDD (PnCDD) isomers were both present as minor constituents (3,8).

2. The pyrolysis (burning) of penta and 2,4,6-trichlorophenate yielded only 1,3,6,8 and 1,3,7,9 TCDD isomers (3,8).

3. Only trace amounts, if any, of 2,3,7,8-TCDD are formed by thermal reactions of the phenoxy acid 2,4,5-T (9).

B. PCDDs and PCDFs in Fly Ash and other Combustion Sources

1. Total amounts of PCDDs and PCDFs in fly ash from a municipal incinerator in Switzerland were found to be 0.2 ppm and 0.1 ppm respectively. At an industrial heating facility, the fly ash contained 0.6 ppm PCDD and 0.2 ppm of PCDF were found. The highly toxic PCDDs (2,3,7,8; 1,2,3,7,8; 1,2,3,6,7,8) were the predominant isomers found (3).

2. A study released by Dow Chemical in 1978 and published in 1980 (10) reported no 2,3,7,8 TCDD (10 ppb detection level) in fly ash from their oil and coal power plant; however, 38 ppb of other TCDD isomers were found at a detection limit of 20 ppb. Dow also reported finding detectable levels of PCDDs from fireplaces, automobile and truck mufflers and cigarette smoke. Dow concluded that dioxins have been present since the advent of fire. The results of their study are presented in Table 1 on page 4.

is sentence
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still considered
overzealous
proximation
Dow.

In contrast to Dow's 1978 report, the authors tempered their remarks in this more recent report by stating that some combustion sources may be sources of airborne and waterborne particulates containing PCDDs. ~~The~~ The formation of PCDDs was attributed partly to the reaction between inorganic chloride and organic material and not due to the condensation of preexisting polychlorinated phenols.

BULL!

3. Kimble and Gross (11), in contrast with Dow Chemical findings, concluded that coal fired power plants were an insignificant source of TCDD.

non-Significant:
max.: 1 ppt TCDD theroetical

TABLE 1

Dow Chemical's Findings on PCDDs and PCDFs
In Fly Ash and Other Combustion Sources (Reference #10)

Sample	Number of samples	Apparent dioxin content, ng/g(ppt					
		TCDD					
		Other isomers	2,3,7,8-	Total	HxCDD	HpCDD	OCDD
Incinerators, powerhouse							
Dow powerhouse	1	38(20)	N.D.(10)	38(20)	2	4	24
Dow rotary incinerator stack (present normal operation with supplemental fuel)	5			N.D.(2)	1-5	4-100	9,950
Dow stationary tar burner stack (normal operation with supplemental fuel)	5	N.D.	N.D.	N.D.	1-20	27-160	190-440
U.S. municipal incinerator (electrostatic precipitator)(Nashville, TN)	1	7.3	0.4	7.7	14	28	30
European municipal incinerators				2-20	30-200	60-130	40-120
Mufflers							
Diesel truck muffler	2	0.02	0.003	0.023	0.020	0.100	0.26
Auto muffler	4	N.D.-0.004	N.D.-0.004	N.D.-0.008	N.D.	0.003-0.01	0.02-0.0
Other sources							
Home fireplace soot	2	N.D.-0.3	N.D.-0.1	N.D.-0.4	0.2-3	0.7-16	0.9-25
Home electrostatic precipitator	1	0.4(0.4)	0.6	1.0	34	430	1,300
Cigarette smoke	2	N.D.	N.D.	N.D.	0.004-0.008	0.009	0.02-0.0
Charcoal-broiled steak	4	N.D.	N.D.	N.D.	N.D.	N.D.	0.03(0.0

Where multiple samples were analyzed from similar sources, the range of observed values is shown. 2) N.D. indicates that the signal observed was less than 2.5 times noise. Limits of detection not within these ranges are shown parenthetically after the value reported for signals between 2.5 and 10 times noise. 3) Data without supplemental fuel is not included here because this practice has been eliminated.

No TCDD was detected at a detection limit of 1.2 ppt in fly ash. The authors concluded that the conversion of less than 1 x 10^{-10} of the chloride in coal would be converted to TCDD. The authors concluded that this reaction would be insignificant.

C. Formation of PCDFs by Pyrolysis of PCBs

1. The pyrolysis of commercial PCBs (Aroclor 1254 and 1260) at temperatures of 500-600°C has been shown by Rappe and his co-workers to yield 30 major and more than 30 minor PCDFs. From 3 to 25% of the pyrolyzed PCB was converted to PCDFs. One of the major constituents was 2,3,7,8-TCDF, the most toxic PCDF. Above 700°C only trace amounts of PCDFs are formed due to the complete destruction of PCB. The formation of PCDFs was shown to be the result of intermolecular cyclizations of PCB (12).

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latter being
e source (precursor)
r the 2,3,7,8-TCDD,
T the PCBs!!

D. Formation of PCDDs by Photolysis

Dechlorination of OCDD in pentachlorophenol will produce tetra, penta, hexa, and hepta chlorinated dioxins. Only the 1,4,6,9-TCDD isomer is produced; the highly toxic 2,3,7,8-TCDD is not (7).

IV. Properties of PCDDs and PCDFs

The majority of information on PCDDs has been confined to the 2,3,7,8 isomer of TCDD. Because of structural similarities, the other PCDDs and PCDFs would be expected to have similar properties. A summary of the properties of TCDD is provided below (2,14).

A. Physical Properties

TCDD is a non-volatile, white crystalline solid with a melting point of 302-305°C. Water solubility is very low, being about 0.2 μg/l at 20°C. The solubility of DDT in water is 1.2 μg/l. The compound binds very strongly to soils such as clays and loams. Temperatures in excess of 500°C are

actually destruction occurs
at 980°C.

required for decomposition. UV light and sunlight have been shown to readily degrade TCDD if the TCDD is not bound to soils. and if there is a proton donor present.

3. Environmental and Biological Properties

TCDD, at concentrations from 1 to 100 ppm, is persistent in the environment with an apparent half life of greater than one year. Because TCDD is highly lipophillic, it readily bioaccumulates. Laboratory aquatic tests with TCDD added to soils indicated bioaccumulation throughout simulated aquatic food chains with bioaccumulation factors as high as 26,000 times. In mammalian studies, TCDD is poorly metabolized and eliminated primarily in the feces.

V. Summary of Toxicological Effects of PCDDs and PCDFs in Animals

Extensive toxicological reviews of the health effects of PCDDs and PCDFs have been presented elsewhere (2,6,7,13,14). A brief summary of the toxicological findings, extracted from these reviews, is presented below.

A. TCDD

1. LD50

The LD50 for 2,3,7,8-TCDD has been shown to be as low as 0.6 µg/kg-body weight (bw) (male guinea pigs) in the animal species tested.

2. LC50

The approximate LC50 for coho salmon exposed for 96 hours to 2,3,4,8-TCDD and kept under observation for 60 days was 0.005 µg/l. Mortality was observed at concentrations as low as 0.056 ng/l.

3. Subchronic and Chronic Studies

Doses as low as 0.1 ng/kg-bw have been shown to cause liver degeneration in rats. Daily oral injestion of 1 µg/kg-bw of 2,3,7,8-TCDD has been reported to be lethal to young rhesus monkeys. Death, physiological effects and hormonal alterations have been noted at dosages as low as 0.01 µg/kg-bw/day in feeding studies involving rhesus monkeys (15). Physiological effects included weight loss, loss of facial hair, gangrenous necrosis of the terminal phalanges, acneform eruptions, anemia, epidermal and internal hemorrhages of major organs, and bone marrow and lymphoid hyperplasia.

4. Fetotoxicity and Teratogenicity

2,3,7,8-TCDD has been shown to be fetotoxic and teratogenic at doses as low as 0.01 µg/kg-bw/day. Embryomortality, cleft palate, and kidney abnormalities have been observed in fetuses of rats, mice, and hamsters. Female Rhesus monkeys have exhibited reduced fertility and abortions at feeding levels of 0.01 µg/kg-bw/day (16).

5. Carcinogenicity

In long-term feeding studies with rats, TCDD has been reported to be carcinogenic at levels as low as ~~0.1~~ 0.01 µg/kg-bw/day.

B. Other Chlorinated Dioxins (PCDDs)

Of the six chlorinated dioxins found in pentachlorophenol, two (1,2,3,7,8,9 and 1,2,3,6,7,8) would be expected to have marked toxicity given that the 2,3,7,8 positions are halogenated (7). In fact, these PCDDs are the only two HxCDDs which have been studied. The LD50 is respectively 60 and 100 µg/kg in guinea pigs. However, only trace levels of the 1,2,3,7,8,9 isomer are found in PCP. Chronic studies have determined that a mixture of these two isomeric dioxins at doses as low as 10 µg/kg/day were highly lethal to rat fetuses during late gestation. Subcutaneous edema of the fetuses were observed at 1 µg/kg/day while a 0.1 µg/kg dose did not yield any fetal anomalies. [illegible]

Studies on the HpCDD of probable highest toxicity (1,2,3,4,6,7,8) with guinea pigs discovered relatively low toxicity of the compound with a single oral dose of 180 µg/kg. Further studies are currently in progress at NIEHS. In the limited studies conducted with OCDD, oral doses of 4 g/kg did not cause death in male mice. In a teratology study, no signs of maternal toxicity or fetal anomalies were observed in rats that received 500 mg/kg-bw/day of OCDD.

C. Polychlorinated Dibenzofurans (PCDFs)

From the limited studies conducted to date on PCDFs which are predicted to have the highest toxicity (2,3,4,7,8 and 1,2,3,6,7,8), the 2,3,4,7,8 dibenzofuran showed significant toxicity. In guinea pigs, 100% mortality was observed with a single oral dose of 10 µg/kg. Enlarged livers were noted upon sacrifice 30 days after a single dose of 1 µg/kg.

VI. Summary of the Toxicological Effects of PCDDs and PCDFs on Humans

A. Human Exposure from Industrial Accidents

There have been a number of industrial accidents during the manufacture of chlorinated phenols that have resulted in human exposure to TCDD (2,13). The more noteworthy are presented below:

1. Nitro, West Virginia (1949). An explosion at a 2,4,5-T chemical plant led to 228 cases of chloracne among exposed workers. Symptoms included shortness of breath, intolerance to cold, palpable and tender liver, loss of sensation in extremities, fatigue, nervousness, irritability, insomnia, loss of libido, and vertigo.

2. Germany, 1953. An accident at a 2,4,5-TCP plant caused 42 persons to have serious dermatitis, 14 of whom developed damage to internal organs, and 7 experienced nervous system disturbances.

3. Germany, 1954. Thirty-one workers at a 2,4,5-T plant suffered from chloracne and experienced various psychological symptoms. Researchers tentatively identified the causative agent as TCDD.

How could they when the compounds was only synthesized in the 1960s by DOW.?

4. United States (1964). At a 2,4-D and 2,4,5-T plant, 11 of 29 workers developed chloracne and porphyria cutanea tarda (liver damage).

5. Derbyshire, England (1968). An explosion at a 2,4,5-TCP plant caused 79 cases of chloracne, many of them severe, among workers.

6. Seveso, Italy (1976). An accident at a chemical plant released approximately 2 to 10 pounds of TCDD over a wide area. Hundreds of animals died, and ~~many~~ 175 area residents (42) ~~(in excess of 100)~~ had confirmed cases of chloracne. Reports of increased spontaneous abortions have not been confirmed. Recent reports have confirmed that cow milk samples collected in 1976 near the explosion area had TCDD levels approaching 8 ppb (17). Rabbit liver samples were found to contain ppb levels of TCDD (18).

B. Human Exposure From Waste Oil and 2,4,5-T

1. Missouri (1971). A major poisoning episode affected humans, horses, and other animals. Waste oil, containing TCDD, had been sprayed on riding arenas to control dust. Within days, birds were found dead on the arena floor. Of 85 horses exercised within the arena, 62 became ill, and 48 died. Horses died over a period of 2 1/2 years. Human symptoms were less severe, but one 6 year old female child, who played in the arena, developed hemorrhagic cystitis.

Analyses of soil samples from the area showed the presence of TCDD ranging from 31.3 to 33.0 ppm.

~~2. Alsea, Oregon (1979). In 1978, the U.S. EPA, in conjunction with local and state health officials, began to investigate spontaneous abortion rates, in response to local complaints. Eight women living in the area claimed to have experienced 10 miscarriages since 1973. The area where the women were located was forested land which had been sprayed for years with 2,4,5-T. A study of six year abortion rates concluded: (1) that women residing in the area where 2,4,5-T usage was extensive, had a statistically higher rate of abortions than than women residing in two control areas (one rural, one urban), (2) that significant correlation existed between the pounds of 2,4,5-T applied and the rate of spontaneous abortion after a lag time of 2 or 3 months; and (3) that there was a statistically significant seasonal cycle in the abortion index, with the highest peaks in June. These findings led to the immediate suspension of 2,4,5-T usage in 1979 (4). Although these data do not prove a cause and effect relationship, they are believed to highly suggestive (40).~~

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3. Australia (1979)

A strong correlation was noted with the use of 2,4,5-T and the frequency of birth defects involving the brain or spinal column (41).

VII. Studies Involving PCDDs and PCDFs Conducted in the Great Lakes Area

A. Dow Chemical Monitoring Study, Midland, Michigan (June, 1978)

In June, 1978, Dow Chemical reported results of their monitoring program to the Michigan Department of Natural Resources, as requested by that agency (19). The data has been summarized on the following page.

1. Tittabawassee River Water and Sediment (1976)

Ten samples (two water and eight sediment) from the Tittabawassee River above and below the plant showed no detectable amounts of TCDD. The detection limit (DL) for sediments ranged from 10 to 50 ppt. Water samples were analyzed at a DL of 1 ppt.

2. Plant Discharge Stream (1976-1978)

Composite and "grab" samples of wastewater discharge revealed no detectable levels of TCDD in 13 of 14 samples (DL 5 ppt or less). One sample was found to contain 8 ppt of TCDD.

3. Bioconcentration Study (1978)

Analyses of caged fish (trout) placed six miles downstream, for periods of 7 to 30 days, revealed no detectable levels of TCDD at a LD of 20 ppt. However, the 30 day trout analyzed with more sensitive methods revealed TCDD at levels ranging from 10 to 20 ppt.

Five of six fish placed in the tertiary pond for 7 days were found to have levels of TCDD ranging from 30 to 50 ppt.

4. Native Fish Studies

1976

TCDD was detected in 4 of 9 catfish samples at levels ranging from 70 to 230 ppt. Three of the four samples in which TCDD was detected were located downstream of Dow's chemical effluent. OCDD was found in 8 of 9 fish, collected both upstream and downstream of Dow's discharge. The levels ranged from 40 to 150 ppt. One fish sample was positive for HxCDD (90 ppt). Detection limits ranged from 10 to 200 ppt.

1977

Fish caught downstream of Dow at Smith's Crossing in the Tittabawassee River revealed contamination in 9 of 14 fish tested. TCDD levels ranged from 20 to 244 ppt.

Saginaw Bay, 1978

Of three fish collected from Saginaw Bay, one (catfish) was found to contain 24 ppt TCDD, at a DL of 10 ppt.

any 2,4,5-TCP?

5. Chlorophenols

Chlorophenols were found in the Dow effluent up to levels of 70 ppb. 2,3,4,6-TCP and PCP were present at ppb levels below Dow's dam sediments of the Tittabawassee River. All the above mentioned chloroophenols were found in fish at ppb levels below Dow's dam. Pentachlorophenol was detected in fish above Dow's waste water stream.

Based upon these findings by Dow Chemical, EPA concluded that widespread environmental contamination of PCDDs existed. Since this represented a substantial risk, EPA requested further refinement of the data supplied to Michigan DNR. EPA also concluded that Dow's discharge represented the major source, if not the only source of TCDD contamination found in the Tittabawassee and Saginaw Rivers and Saginaw Bay in Michigan (20,21).

B. Dow Chemical Monitoring Study, Midland, Michigan (October, 1978)

Results of PCDD sampling conducted on Dow's waste streams indicated the presence of 2,3,7,8-TCDD in the 2,4,5-T sludge at 10 ppt and in trichlorophenol scrubber fluid at 20 ppt (22). The 2,4,5-T sludge also contained 11 ppt HxCDD, 4 ppt HpCDD, and 43 ppt OCDD. Brine wastes and sludges from 2,4-D processes were negative for PCDDs.

WAY:
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CP sludge.
evels is
HxCDD,
OCDD and
DD in
.,5-T
cess waste.

C. U.S. EPA TCDD Fish Monitoring Program (December, 1978)

In response to Dow Chemical's monitoring report, which indicated incidental contamination of fish from the lower Tittabawassee River, fish were collected and analyzed from the Tittabawassee, Grand and Saginaw Rivers (23). Twenty-six out of 35 fish samples contained detectable levels of TCDD ranging from 4 to 695 ppt. Eleven of these fish samples contained TCDD levels greater than 40 ppt. All but two of these samples were from locations downstream of Dow Chemical Company. Four out of 6 samples collected upstream were found to be negative for TCDD. Data is presented in Table 2 on pages 14 and 15. Analyses of blind controls, which consisted of various known levels of TCDD, indicated that the positive results may be viewed with confidence and within 20% of the values reported. The analytical method utilized did not distinguish between isomeric forms of TCDD. Three other isomeric forms of TCDD, besides the highly toxic 2,3,7,8 were noted.

Based upon a review of these findings, the FDA advised a tolerance limit of 100 ppt in fish. Where PCDDs had been found in fish below 100 ppt, it was recommended that no more than one average size portion of fish be consumed per week (24).

D. U.S. EPA Monitoring Study of Dow Chemical, Midland, Michigan (April, 1979)

Results of TCDD analysis of samples taken from Dow's 2,4,5-Trichlorophenol injection well (60 x 10^{-15} detection limit), phenol treatment system (82 ppt DL), Dow's effluent (2 x 10^{-15} DL), and upstream Tittabawassee River (50 x 10^{-18} DL) were found to be negative (25). However, the carbon

TABLE 2 -

RESULTS OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN (TCDD) FISH MONITORING PROGRAM CONDUCTED BY THE U.S. ENVIRONMENTAL PROTECTION AGENCY IN MICHIGAN DURING 1978

Tittabawassee River				
Tittabawassee Road	Carp	42.2	52	8
	Yellow Perch	16.2 11.0b	20	8
	Carp	39.7	93	6
Freeland Road	Carp	40.0	32	4
	Yellow Perch	16.8 12.3	10	5
Smith's Crossing Road	Channel Catfish	50.3	273	6
	Carp	38.0	22	11
	Channel Catfish	38.2	695	60
	Carp	23.1	49	1
	Sucker	35.6	8	4
	Sucker	38.1	21	4
Above Dow Dam	Channel Catfish	30.9	42	9
	Carp	34.6	NDc	5
	Carp	33.0	ND	9
	Channel Catfish	38.1	28	2
	Yellow Perch	19.3	ND	4
Dublin Road	Carp	52.2	ND	9

TABLE 2 (cont'd)

Saginaw River				
Wickes Park	Carp	46.0	62	13
	Yellow Perch	11.9 11.4	ND	11
Block's Marina	Channel Catfish	69.0	105	7
	Channel Catfish	48.5	52	8
	Carp	51.8	28	4
Mouth	Channel Catfish	46.0	30	6
	Carp	45.0	153	13
	Yellow Perch	16.0 16.0	11	4
Grand River				
Jones Road near Eagle	Carp	39.0	20	4
	Carp	39.0	41	8
	Channel Catfish	----	29	6
	Smallmouth Bass	35	7	6
	Smallmouth Bass	38.0	8	6
Near Ionia	Carp	50.8	ND	2
	Sucker	48.3	ND	2
Saginaw Bay				
Lat. 43 40' 16"	Sucker	28.2 31.0	4	2
Long. 83 50' 21"	Yellow Perch	24.2 26.8	ND	3

TABLE 2 (cont'd)

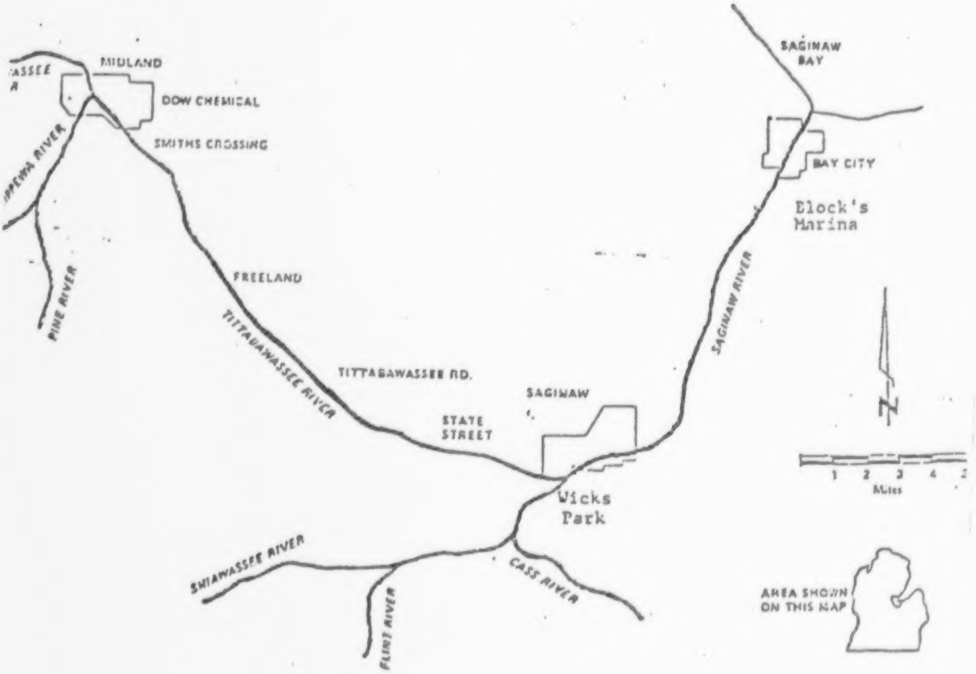
Location	Fish Species	Fish Length (cm)	TCDD Level (ppt)a	TCDD Dectection Limits (ppt)
Lake Michigan				
Off Saugatuck	Lake Trout	50.8	ND	7

a ppt indicates parts per trillion which is equivalent to ng/kg.

b signifies composite fish sample.

c ND indicates that TCDD was not detected in the fish sample at the corresponding detection limit.

SAGINAW RIVER BASIN


ASSEE R
MIDLAND
DOW CHEMICAL
SMITHS CROSSING
IPPEWA RIVER
PINE RIVER
FREELAND
TITTABAWASSEE RIVER
TITTABAWASSEE RD.
STATE STREET
SAGINAW
Wicks Park
SAGINAW BAY
BAY CITY
Elock's Marina
SAGINAW RIVER
N
1 2 3 4 5
Miles
SHIAWASSEE RIVER
FLINT RIVER
CASS RIVER
AREA SHOWN ON THIS MAP

filter concentration system utilized was believed to be unsuitable for concentrating contaminants water samples.

E. Composite Fish Sample from the Tittabawassee River (December, 1980)

A single composite fish sample of five carp, supplied by the MDNR, (collected downstream of Dow Chemical) and analyzed by Columbia National Pesticide Laboratories, yielded positive results for various PCDDs and PCDF's. The 2,3,7,8 isomer of TCDD predominated (26).

The results are shown below:

	ppt Detection Level
TCDD	81
PCDD	31
HxCDD	44
HpCDD	53
OCDD	14
Total PCDD	223

	ppt Detection Level
TCDF	37
PCDF	73
HxCDF	145
HpCDF	31
OCDF	4
Total PCDF	290

F. Dow Chemical Monitoring Study, Midland, Michigan (October, 1980)

In October, 1980, Dow Chemical published a report on environmental sampling for chlorinated dioxins (10). Included in this report were data from analyses of soils, dusts, and emissions from a rotary kiln incinerator, collected between 1976 and 1978. These sites were located within the Dow Chemical facility. The results, presented in Table 3 on page 20, (given in ppb) clearly shows that the highest levels of PCDDs in soil and dust samples were those collected at Dow Chemical. One soil sample was found to contain 100 ppb of 2,3,7,8-TCDD. OCDD was found at levels as great as 20,000 ppb. Particulate matter collected from a rotary kiln incinerator, operated without supplementary fuel and burning wastes from the facility, had levels of total TCDD reaching 14,000 ppb (see Table 4 on page 18). Mention is made that results from the particulate analyses

They were burning 2,4,5-TCP
still bottoms.

Particulates collected from the scrubber water contained TCDD at levels up to 2,500 ppb when the kiln was operated without supplementary fuel (see Table 5 on page 19).

G. Canadian Study on Gull Eggs from the Great Lakes

Dr. Douglass Hallet of the Canadian Wildlife Service reported finding 2,3,7,8-TCDD in Herring gull eggs, collected in 1980, at 12 different locations within the Great Lakes (27). The highest levels of TCDD were found in Saginaw Bay (43-90 ppt). Levels in Lake Ontario eggs were slightly lower and ranged from 44-64 ppt. Trace levels (approximately 3-10 ppt) were found in eggs collected at other Great Lakes sites. There have been no reports of abnormalities in gull chicks or mature birds. The 2,3,7,8-TCDD isomer was the only one detected in Herring gull egg samples at all locations. Egg samples collected from Lake Ontario in 1971 had TCDD levels of 800 ppt. These results indicate a decrease in TCDD levels and may be the result of Hooker Chemical discontinuing production of 2,4,5-Trichlorophenol in 1971. Confirmational analyses of samples are now underway in U.S. laboratories. Preliminary analyses from the University of Nebraska indicates that total TCDD results are about 10 fold greater than those found in the Canadian study, probably due to the use of a different recovery method and reporting the data corrected for losses during recovery (28).

H. Gull Flesh Analyses From the Great Lakes (December, 1980)

Two flesh samples from gulls in the Saginaw Bay were recently analyzed by Dr. David Stalling of the Columbia National Fisheries Research Laboratory (19). In the two samples, the 2,3,7,8 TCDD isomer predominated and was

Chlorinated Dioxins in the Environment (Reference #10)

		Apparent dioxin content, ng/g (ppb)[1]					
	Number of samples	TCDD					
		Other isomers	2,3,7,8-	Total	HCDD	H CDD	OCDD
(Gaylord, MI)	5			N.D.[2]	N.D.	N.D.-0.05	N.D.-0.2
(Lansing & st Lansing, MI)	5			N.D.	0.03-1.2	0.03-2	0.05-2
metropolitan hicago, IL)	8			0.005-0.03	0.03-0.3	0.1-3	0.4-22
hemical idland, MI)	5	0.8-18	0.3-100	1-120	7-280	70-3,200	490-20,000
hemical lab.	6	0.5-2	0.7-3	1-4	9-35	140-1,200	650-7,500
nd, Michigan	2			0.03-0.04(0.02)[3]	0.2-0.4	2-4	20-30
politan etroit, MI)	4			N.D.-(0.03)	N.D.-0.3	0.3-4	0.1-4
politan t. Louis, MO)	1	0.16	0.12	0.3	2	34	210
politan hicago, IL)	2			0.04(0.04)	N.D.-0.3	0.6-3	3-8

ere multiple samples were analyzed from similar sources, the range of observed values shown.

D. = Not Detectable

lue in parentheses indicates detection limit.

Chlorinated Dioxin Content of Particulate Matter
From the Rotary Kiln Incinerator
Reference #10

These data indicate that Dow burned (some) 2,4,5-TCP and PCP still bottoms

	Fuel[1]		Apparent dioxins, ng/g (ppb)				
			TCDD				
Sample	Kiln	Secondary	Other isomers	2,3,7,8-	HCDD	H_7CDD	OCDD
			Without supplemental fuel				
R_1F_1	T,SW,G	T	1,800	2,800[2]	13,000	110,000	180,000
R_1F_2	T,SW,G		5,000	8,200[2]	65,000	510,000	810,000
R_2F_1	T,SW,G		3,300	110	1,300	2,000	3,000
R_3F_1	T,SW,G		12,000	N.D.(260)[3]	5,600	37,000	59,000
			With supplemental fuel				
R_3	T,SW,G	O,G	N.D.(8.0)	N.D.(2.0)	1.4	13.0	30.0
R_4	T,SW,G		N.D.(7.0)	N.D.(5.0)	N.D.(1.0)	4.0	9.0
R_5	T,SW,G		N.D.(2.0)	N.D.(2.0)	N.D.(0.5)	6.0	15.0
R_6	T,SW,G	T,G	N.D.(2.0)	N.D.(4.0)	5.0	27.0	170.0
R_7	T,SW,G		N.D.(2.0)	N.D.(2.0)	4.0	110.0	950.0

1. T, tars; SW, solid waste; G, gas; O, oil.

2. The high results reported for the 2,3,7,8-isomer are probably due to analysis by the nonspecific GC-MS packed-column method. Later results were obtained with a capillary column specific for 2,3,7,8-TCDD. The TCDD results from run 1 (R_1) are not comparable to those from runs 2 and 3.

3. Numbers in parentheses are limits of detection.

TABLE 5

Chlorinated Dioxins in Rotary Kiln Incinerator Scrubber Water

Reference #10

Sample	Apparent dioxins, ng/g (ppb)				
	TCDD				
	Other isomers	2,3,7,8-	HCDD	H_7CDD	OCDD
articulates filtered from scrubber water					
ithout supplemental fuel	300	2,200*	3,400	26,000	42,000
ith supplemental fuel	14	32*	200	970	1,200
iltered scrubber water without supplemental fuel	0.0018(0.01)	0.001*(0.0006)	0.005	0.024	0.026

The analytical method did not separate 2,3,7,8-TCDD from 11 other isomers.

found at levels of 70 and 160 ppt. HxCDD-and OCDD were also found resulting in a total PCDD level of 135 and 196 ppt. Total PCDFs were found at 117 and 121 ppt with the PnCDF and HxCDFs being the predominate furans. The 2,3,7,3--TCDD levels have been confirmed within experimental error by the U.S. EPA Research Triangle Park laboratory and by Buser of Sweden.

I. PCDDs Analyses on Great Lakes Fish (April, 1930)

The Columbia National Fisheries Research Laboratory has obtained preliminary, positive results for TCDD, on composite fish samples from the Great Lakes (30). This fish sampling is being conducted under the National Pesticides Monitoring Program. Both Huron and Ontario fish range in TCDD concentrations from 33 to 160 ppt. The highest values were reported to fish collected from Saginaw Bay. TCDD was not detected in fish from Erie and Lake Superior. The predominate isomeric form of TCDD discovered has been 2,3,7,8-TCDD. Data collected to date is presented in Table 6, pages 24 and 25. The New York State Department of Health is currently screening Lake Ontario fish for TCDD contamination. Previous analyses conducted by the agency determined TCDD contamination at about 10 ppt. Fish samples collected below the Niagra River had no detectable levels of TCDD. Sediment samples taken from streams in the vicinity of Hooker Chemical, where 2,4,5-TCP wastes were dumped, have been reported to be positive for TCDD (31).

J. NIEHS Analyses of a Michigan Dairy Herd Tissue Samples and Michigan's Study on Pentachlorophenol Treated Wood (1978-1979)

Studies by NIEHS of a dairy herd in Michigan, presumed to be contaminated by contact with PCP treated wood, revealed fat and liver contamination, respectively, with OCDD at 7 and 15 ppb; HpCDD at 6 and 15 ppb; and HxCDD

TABLE 6

Residues of Dibenzofurans and Dibenzo-p-dioxins in Composite Samples of Fish and Gulls From Different Sites on the Great Lakes. Residues are in parts-per-trillion and by Number of Chlorine Substitutions.

Site and Sample	Dibenzofurans						Dibenzo-p-Dioxins						Sample Index No.
	4Cl	5Cl	6Cl	7Cl	8Cl	Total	4Cl	5Cl	6Cl	7Cl	8Cl	Total	
Saginaw Bay, L. Huron Carp	27	44	34	44	4	153	94	157	122	12	ND	385	GL1
Saginaw Bay, L. Huron Herring Gull #1	16	28	57	17	3	121	160	ND	20	ND	19	199	GL2
Saginaw Bay, L. Huron Herring Gull #2	15	50	40	7	5	117	70	ND	88	ND	28	186	GL3
Bay Port, L. Huron Carp	5	12	5	6	1	27	27	21	ND	31	32	111	GL7
L. Huron 79-06 (Duplicate analyses) Lake Trout (1)	32	27.5	16.3	8.7	6.3	91							GL13
(2)	19	29	16	4	2	70							
Tittabawassee River-below Dow Dam Carp	37	73	145	31	4	290	81	31	44	53	14	223	GL9
L. St. Clair 79-12 Walleye	4	4.2	0.7	0.7	2.6	12							GL14
L. Erie 79-09 Walleye	17.6	9.2	5.7	5.3	2.2	40							GL15
Port Clinton, L. Erie Carp	5	5	2	4	1.5	17.5	ND	9	ND	11	30	50	GL8
Saugatuck, L. Michigan Lake Trout	35	41	8	1.4	1.4	87	5	ND	ND	ND	ND	5	GL4

TABLE 6 (Continued)

Residues of Dibenzofurans and Dibenzo-p-dioxins in Composite Samples of Fish and Culls From Different Sites on the Great Lakes. Residues are in parts-per-trillion and by Number of Chlorine Substitutions

Site and Sample	Dibenzofurans						Dibenzo-p-Dioxins						Sample Index No.
	4Cl	5Cl	6Cl	7Cl	8Cl	Total	4Cl	5Cl	6Cl	7Cl	8Cl	Total	
L. Michigan-79-check Lake Trout	33	61	10.4	4.4	2	110							GL16
Keweenaw, L. Superior Bloater Chub	26	9	2	1	2	40	ND	ND	ND	ND	ND	ND	GL5
Isle Royale, L. Siskiwit, L. Superior Lake Trout	10	5	ND	ND	Trace	15	ND	ND	ND	ND	Trace	Trace	GL10
L. Superior 79-02 Lake Trout	19	8.8	5.4	3.7	2.7	40							GL12
Roosevelt Beach, L. Ontario, Brook Trout	19	4	3	3	3	32	33	ND	ND	ND	ND	33	GL6
L. Ontario 79-10 Lake Trout	34	48	29	6	2	119							GL11

at 1 and 9 ppb (7). The presence of these contaminants was suspected of causing the illnesses noted in the cattle. Analyses conducted for PCDDs, by the State of Michigan, on the PCP treated wood in barns established the link between the wood and the presence of PCDDs in the cattle samples (32). OCDD ranged from 289-5000 ppb, HpCDDs from 9-1161 ppb, and HxCDDs from non-detectable to 3.7 ppb in the wood.

K. U.S. EPA Assessment of Environmental Contamination in the Area of Hemlock Michigan (October, 1980)

As a result of numerous health complaints involving humans and domestic animals, the U.S. EPA conducted a major environmental assessment of the Hemlock, Michigan area during 1980 (33). Citizens were dissatisfied with previous studies (negative findings) conducted by county and state agencies. Residents were concerned that potable groundwater had been contaminated from a brine reinjection system operated by Dow Chemical in that area.

An extensive study of TCDD in environmental and biological samples yielded no positive results at a detection limit of low ppt levels. Downspout soils, house dusts, a well sediment sample, a septic tank sludge, and sediment samples from Dow's injection systems were also analyzed.

L. Crete Metals, Crete, Illinois (December, 1980)

TCDD and TCDF analyses were performed in 1980 on environmental and biological samples as a result of health complaints and the death of several horses near a wire reclamation facility (34). In the reclamation process, insulating and other materials were burned off wires, x-ray plates and transformer cables and other unknown metal-containing products. The samples analyzed and results obtained are presented below in Table 7.

TABLE 7

	TCDD (ppt)	TCDF (ppt)
Stack #2 (Scrap~~p~~ing)	410	11,600
Furnace #2 (Scrap~~p~~ing)	58	730
Soil (on site)	21	230
Adipose (dead horse)	45	165

A variety of isomeric forms of TCDD and TCDF were detected, however, the analytical technique utilized could not adequately discriminate and quantify the isomers.

M. Dow Chemical Study of Wives of 2,4,5-T Workers (January, 1981)

No significant differences in miscarriages, stillbirths, infant deaths, or malformations were noted between wives of 2,4,5-T workers and unexposed workers (35). Other factors, such as the mother's age, complications during delivery, smoking, and mother's occupation, did not reveal any links with reproductive anomalies and exposure to 2,4,5-T. In the unexposed group, Dow reported 2031 conceptions compared to 737 in the control group, suggesting that 2,4,5-T exposure may affect fertility.

VIII. Federal Regulations on PCDDs and PCDFs

Except for disposal requirements for wastes containing 2,3,7,8-TCDD, no other regulations exist for PCDDs and PCDFs (36). Not considering carcinogenicity, the acceptable daily intake (ADI) for TCDD has been calculated at 10^{-4} μg/kg/day (37). Based upon Dow's two year study on rats, the ADI should be 10^{-5} μg/kg/day (38). At this level, a 154 pound person could only consume 1/2 pound of fish per week with 22 ppt TCDD, considerably lower than the advisory limit of 100 ppt set by the FDA. The draft ambient water quality criteria is 4.55×10^{-8} μg/1 to reduce the lifetime human carcinogenic risk to 10^{-6} (14). If this criteria would be extrapolated to fish, not even one kg of fish (2.2 pounds) containing 1 ppt 2,3,7,8-TCDD could be consumed during a lifetime to keep the risk at 10^{-6} for cancer. No criteria for aquatic wildlife have been drafted.

interesting case presented here.

IX. Conclusions

1. Major environmental contamination with PCDDs and PCDFs. particularly with 2,3,7,8-TCDD isomer is due to the release of commercial products containing these contaminants and secondarily from the combustion of certain chlorophenolic or similar compounds (~~i.e., PCBs~~).

please discriminate here – it is NOT the PCBs but the tetrachlorobenzene present in the formulation only the 1,2,4,5-tetrachlorobenzene on condensation under appropriate conditions to 2,3,7,8-T

2. Other combustion sources, such as automobiles and coal fired power plants, appear to be insignificant sources of PCDDs and PCDFs.

orrect

3. Dow Chemical of Midland, Michigan, has extensively contaminated their facility with PCDDs and PCDFs and has been the primary contributor to contamination of the Tittabawassee and Saginaw Rivers and Lake Huron.

YES

4. The differential contamination of biological samples from the Great Lakes, being largely confined to Lake Huron and Ontario, suggests that industrial contamination is responsible.

OOD
UCK

5. The consumption of fish from the Tittabawassee River, the Saginaw River, Saginaw Bay, and possibly other sites in the Great Lakes should be prohibited.

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31. Eposito, M. P. and D. R. Watkins, "Airborne Dioxins: The Problem in Review," Paper presented at the 73rd Annual Meeting of the Air Pollution Control Association, June 22-27, 1980.

32. August 13, 1978 Communication to G. Marsh, Pesticides Branch, EPA Region V, from Thomas O. Tiernan, Wright State University.

EPA - Dr Paul Brown

Ban fish containing dioxin, report on Great Lakes urges

By MICHAEL KEATING
and RAE TYSON

A confidential U.S. report says that consumption of some Great Lakes fish containing the chemical dioxin should be prohibited because of the increased risk of cancer to those eating them.

Dioxin, in its most toxic 2,3,7,8-TCDD form, is more deadly than strychnine or cyanide and is believed to be the most poisonous chemical made by man.

In tiny amounts, dioxin causes painful chloracne (large cankers in soft tissue) in humans. It is strongly suspected as a human carcinogen. Test animals fed amounts in the parts per billion and trillion range showed such effects as cancer, birth defects, liver degeneration, hormone changes and death.

The report, prepared by a group within the U.S. Environmental Protection Agency, says levels of dioxin as high as 696 parts per trillion have been discovered in fish taken from the Great Lakes.

In recent years, Canada and the International Joint Commission on water quality have said that food and water should be completely free of dioxin. In an interview yesterday, a U.S. Food and Drug Administration official advised against eating more than one fish a week containing more than 100 parts per trillion of dioxin. Only limited amounts of fish containing 50-100 ppt should be eaten, he added.

Aside from details in the leaked report and one or two preliminary studies, there is no way the public can find out how much dioxin there is in fish they are eating.

Government officials in both Canada and the United States say a detailed report on dioxin in Great Lakes fish will be ready for release about the end of this month.

Still in draft form, the EPA's dioxin report obtained by The Globe and Mail and the Niagara Gazette in Niagara Falls, N.Y., says: "The consumption of fish from the Tittabawassee River, the Saginaw River, Saginaw Bay (all in Eastern Michigan) and possibly other sites in the Great Lakes should be prohibited."

One researcher said the report was leaked because "otherwise it probably would not see the light of day." This was a reference to the change to administration in Washington.

The report says that Dow Chemical Co. of Midland, Mich., has extensively contaminated its chemical factory with dioxins and the chemically related dibenzofurans "and has been the primary contributor to contamination of the Tittabawassee and Saginaw Rivers and Lake Huron."

The report says that less is known about the effects of dibenzofurans, but they can be created by low-temperature burning of such chemicals as polychlorinated biphenyls (PCBs) and appear to be very toxic.

It says that based on Dow's own two-year study of the effects of dioxin on rats, "a 154-pound (70-kilogram) person could only consume half a pound (225 grams) of fish per week with 22 parts per trillion of TCDD (dioxin.)" Going by a draft U.S. water quality guideline on cancer risk, it would be unsafe to eat any fish contaminated with dioxin, the EPA study adds.

The definitive report on dioxin in fish is being prepared by five main and six advisory research groups in Canada and the United States. They have analyzed between 250 and 300 fish samples from the Great Lakes, mainly Lake Ontario, and are now rechecking their results for accuracy.

In an interview yesterday, Douglas Hallett, a senior Canadian researcher, said the report, which should be made public during an international meeting by the end of this month, will tell how much dioxin is in the fish and will give a health warning. The risk to those eating the fish will vary because "some people are genetically predisposed to handle chemicals" and some are not.

Mr. Hallett, who calls dioxin "a bloody hazardous compound," drew attention to the contamination situation last fall when he found that herring gull eggs from around Lake Ontario were contaminated with the chemical.

He traced the poison in the eggs back to production of dioxin-contaminated trichlorophenols, particularly by Hooker Chemicals and Plastics Corp. in Niagara Falls, N.Y. In 1974, Hooker stopped producing TCP and since then dioxin levels in gull eggs have dropped by a factor of about ten.

Some dioxin is still leaking into the Niagara River and Lake Ontario from chemical waste dumps such as Love Canal and Hyde Park on the Niagara frontier. Mr. Hallett said that after the dioxin figures are released this summer governments will have to decide whether to ban or restrict fishing. Ontario estimates the commercial and sports fisheries on Lake Ontario are worth more than $20-million a year and those in Lake Huron at least as much.

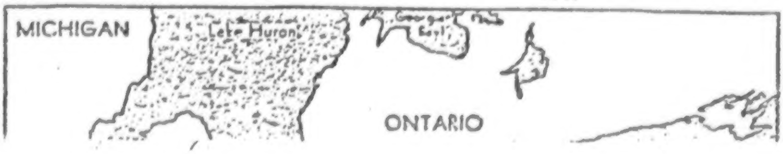
On Wednesday, Ontario Environment Minister Keith Norton was quoted as saying dioxin was found in fish in all the Great Lakes "but there is nothing to be alarmed about." The next day he told reporters that he must have misunderstood the question and meant to say there were tests of fish from all the lakes but he saw dioxin figures only for some Lake Ontario fish.

When shown a copy of the confidential U.S. report, he said the conclusions "raise some very real concerns. It may mean some restrictions if the standards quoted are accepted."

Looking at a copy of Ontario's report, stamped "confidential," he said its readings for Lake Ontario fish were not as high as the U.S. figures. Mr. Norton said no Ontario fish contained more than 22 parts per trillion of dioxin.

The U.S. study found levels up to 696 ppt in fish downstream from Dow's Midland plant. "Both Lake Huron and Ontario fish range in dioxin concentrations from 20 to 160 ppt. The highest values were reported from fish from Saginaw Bay."

Michael Keating is environmental reporter for The Globe and Mail. Rae Tyson is environmental reporter for the Niagara Gazette in Niagara Falls, N.Y.



HAZARDOUS WASTE CONTAMINATION OF WATER RESOURCES

(EPA Implementation of the Superfund Program and Lead Pollution Problems in Dallas, TX)

THURSDAY, MARCH 24, 1983

House of Representatives,
Subcommittee on Investigations and Oversight,
Committee on Public Works and Transportation,
Washington, DC.

The subcommittee met, pursuant to call, at 9:42 a.m., in room 2167, Rayburn House Office Building, Hon. Elliott H. Levitas, (chairman of the subcommittee) presiding.

Mr. Levitas. The subcommittee will please come to order.

The hearings today are a continuation of the oversight investigation which this subcommittee has undertaken with respect to the Superfund Program and the hazardous waste clean up efforts of the Environmental Protection Agency.

Before I get into any specifics on today's hearings, I think there are two or three matters that deserve specific attention.

The first is that yesterday I received and executed an agreement with the counsel at the White House, Mr. Fielding, and Deputy Attorney General Ed Schmults, concerning additional arrangements with respect to the receipt of documents by this subcommittee.

The arrangements as now in place will provide not only that the subcommittee will have full access and receive the documents that we have asked for, but in addition to that, the original position which I had set forth back in September of last year has now been acknowledged.

Our committee staff will be able to have full and free access to the files of the Environmental Protection Agency at the places where those files are located, and be able to go through those files in order to identify the documents that this subcommittee needs.

I am very pleased that that agreement has been made. It is also my understanding that either today or tomorrow we will receive the first set of six site files that the subcommittee requested, and we will be able to get on with that.

The second point I would like to make is that in front of each member is a copy of a letter I received yesterday from Mr. Perry, the General Counsel of the Environmental Protection Agency.

Mr. Snyder. I don't think we have a copy of that.

Mr. Levitas. You do now.

The letter from Mr. Perry relates to the fact that they have become aware that the Environmental Protection Agency had a contract with TRW, Inc., to investigate the cause of environmental and health damage at 15 selected sites.

The results of the contractor's investigation were to be used in evaluating the need for additional or different Resource Conservation Recovery Act regulations. One of the sites involved is the Stringfellow acid pits site in Riverside, CA.

TRW is, it so happens, a contributing generator or responsible party and a potential defendant in that matter. Accordingly, the officials of the Environmental Protection Agency, specifically Mr. Michael Brown, who is Enforcement Counsel, has recommended that TRW be instructed to cease temporarily all work under the contract, and with respect to the Stringfellow site, TRW should be permanently estopped from further work.

I think that we are going to have to get into this a little bit further at a proper time. I am getting additional information on it.

We have received this morning another memorandum pertaining to this, which I have not yet had an opportunity to study in detail. I hope to do so during the day and will make a further report on it to the committee.

Without objection, the agreement between myself, Mr. Fielding, Mr. Schmults that I just referred to will be made a part of the record.

[The information follows:]

THE WHITE HOUSE
WASHINGTON

March 23, 1983

Dear Mr. Chairman:

Following up on our meeting of yesterday, enclosed please find the amended Memorandum of Understanding dated March 22, 1983, which has been executed by Edward C. Schmults and by me.

I am also enclosing a letter to you at Ed's request; I understand that he is going to call you to explain the basis for this letter.

I trust that this Memorandum reflects our discussions and understandings, and is acceptable to you.

Upon execution, I would appreciate receiving a copy for our files.

Sincerely,

Fred F. Fielding
Counsel to the President

The Honorable Elliott H. Levitas
Chairman, Subcommittee on
Investigations and Oversight
B-376 Rayburn Building
Washington, D.C. 20515

MEMORANDUM OF UNDERSTANDING

1. The Subcommittee on Investigations and Oversight ("Subcommittee") of the House Committee on Public Works and Transportation ("Committee") will obtain access as provided herein to all documents in Environmental Protection Agency ("EPA") files relating to Superfund sites selected by the Subcommittee for review.

2. The Subcommittee, or the Subcommittee through its investigators, will initially designate a reasonable number of National Priority List sites which are the subject of its official inquiries undertaken pursuant to its investigation and oversight responsibilities. Additional sites may be designated from time to time by the Subcommittee, or the Subcommittee through its investigators. Reasonable advance notice will be provided to the Associate Administrator for Legal and Enforcement Counsel to permit the Agency to review its files and identify those documents to be designated as "enforcement sensitive."

3. Such access to files for review by the Subcommittee or its investigators shall be either at EPA or by delivery of such files to the Subcommittee.

4. During its review, the Subcommittee investigators may request executive branch staff to be present to assist the investigators in their review of the documents. The Subcommittee investigators will be free to obtain copies of all documents in the files, subject to the conditions of paragraph 5, and to otherwise conduct investigations in an appropriate and customary manner. The investigators shall provide EPA with a record of all documents copied pursuant to this Memorandum of Understanding.

5. The EPA shall identify at the time of copying or delivery any document or portion thereof that is "enforcement sensitive." The designation of "enforcement sensitive" shall be used to identify any document or portion thereof the public disclosure of which could jeopardize the ability of the Executive Branch to pursue any enforcement action.

6. The Subcommittee agrees to afford any document or portion thereof designated as "enforcement sensitive" the confidential treatment of executive session materials. If the Subcommittee votes to release or use in public session any document or portion thereof designated as "enforcement sensitive", reasonable advance notice with opportunity for consultation shall be given to the EPA. Every effort should be made to reach agreement as to whether or not the document should be released or used in public session. If agreement cannot be reached, such documents shall not be released or used in public session without the concurrence of the Chairman and Ranking Minority Member of the Subcommittee. In the event such concurrence cannot be reached, such documents shall be released or used in public session only upon a further vote of the members of the Subcommittee. The documents shall be kept in a locked safe with sign-in procedures for those obtaining access. Staff access shall be limited to those individuals designated jointly by the Chairman and Ranking Minority Member.

7. Nothing in this memorandum shall constitute a waiver of Executive Privilege with regard to any document. The Subcommittee does not acknowledge the applicability of Executive Privilege to any documents subpoenaed.

8. Title to "enforcement sensitive" documents shall remain in the EPA and shall not transfer to the Subcommittee. Upon notice by the Subcommittee to the EPA that the documents are no longer needed, the EPA may retrieve them.

9. This memorandum is intended to solely resolve the issues relative to the subpoena issued by the Subcommittee on Investigations and Oversight of the House vote on December 16, 1982, finding EPA Administrator Anne M. Gorsuch in contempt of Congress and does not bind, or otherwise affect the rights of any other subcommittee of the House with respect to any subpoena which has been or may be authorized. It supercedes and amends the Memorandum of Understanding dated February 18, 1983, between the parties.

10. Following satisfactory implementation of the procedures set forth in the Memorandum of Understanding dated February 18, 1983, as amended herein, the Chairman of the Subcommittee represents that he will undertake and use his best efforts to introduce an appropriate resolution, for consideration by the Committee and the House of Representatives. An essential and nonseparable

condition of this agreement is that the House of Representatives will adopt a resolution containing recitals that implementation of the procedures established herein constitutes a purging of the aforesaid resolution of the House of Representatives of December 16, 1982, certifying that compliance with the Subcommittee's subpoena has been effected; that the House finds no reason for further proceedings; and providing for the Speaker to certify all of the foregoing to the United States Attorney.

Elliott H. Levitas, Chairman
Subcommittee on Investigations
and Oversight
Committee on Public Works
and Transportation

Fred F. Fielding
Counsel to the President

Edward C. Schmults
Deputy Attorney General
Department of Justice

Date: March 22, 1983

Committee on Public Works and Transportation

Congress of the United States
House of Representatives
Room 2165, Rayburn House Office Building
Washington, D.C. 20515
Telephone: Area Code [illegible]

March 21, 1983

Fred F. Fielding, Esq.
Counsel to the President
The White House
Wasnington, D. C.

Dear Mr. Fielding:

I have read the revised Memorandum of Understanding which you enclosed with your letter of March 18, 1983, and made some changes to that version which I feel more accurately incorporate the provisions of the agreement reached with Mr. Dingell.

These changes are consistent with my understanding and reading of the Dingell Agreement. I am anxious to implement what I said I would undertake to do with regard to the contempt citation when we reached agreement on the February 18, 1983 Memorandum of Understanding.

One additional change I have made is to include Ed Schmults as a party to the Memorandum for two reasons. One is that he was a party to the February 18 agreement, and as such should be included in any amendment of that agreement. The second is that Jim Baker had asked me to work with Ed Schmults on this matter.

If you have any questions as to the revisions which I have made, please let me know as soon as possible. If they are agreeable to you and to Ed Schmults, I would appreciate receiving an executed copy from each of you for our files.

With best wishes, I am

Very truly yours,

Elliott H. Levitas
Chairman
Subcommittee on
Investigations and Oversight

Enclosure
cc: Hon. Edward C. Schmults
Deputy Attorney General
U.S. Department of Justice
(with enclosure)

Mr. LEVITAS. Copies will be made available to the members of the subcommittee.

One last point. I want to note that since our last meeting, Mr. William Ruckelshaus has been designated by President Reagan to be Administrator of the Environmental Protection Agency. I don't know Mr. Ruckleshaus personally. I know that he comes with good credentials from his prior services, both as the Deputy Attorney General and as the first Administrator of the Environmental Protection Agency.

I hope that he is given the tools and the mandate to work with in order to change the direction in which the Environmental Protection Agency has been headed, and that many of the problems this subcommittee has encountered hopefully will be removed.

I think that the investigation of our subcommittee which will be ongoing can be of assistance to Mr. Ruckleshaus in finding out many of the types of problems that need to be changed in order to get the Agency back on track.

Those are the only comments that I have at this point, and I will recognize the gentleman from New York, Mr. Molinari.

Mr. MOLINARI. Thank you, Mr. Chairman.

I also want to join in your statement as to the new Administrator. He has excellent credentials. He is off to a very good start.

I think he is going to quickly start to restore confidence in the Environmental Protection Agency. I think we will be able to work with him toward that goal.

Mr. Chairman, for the record, I sent a letter to the Speaker asking whether we could now proceed with a resolution purging Mrs. Burford of contempt. I received a response from him last week saying that it would depend upon yourself, and that you would make the decision when you were satisfied.

In light of the document that you referred to before and the agreement that has now been reached with Counsel to the White House, are you prepared to say whether we are going to be able to move to purge Mrs. Burford of that contempt citation?

Mr. LEVITAS. I thank my colleague for the question. It is my hope that based on this agreement, we will receive the documents that we need.

I have said previously that the performance, not the promise, will be the test. Hopefully, between now and the time we return from the district work period, our staff will have had compliance with this agreement.

We have had too many agreements with the Agency in the past for me to operate solely on the basis of a promise. But certainly, if they are forthcoming and this agreement is implemented, that when we return, I would hope to be able to live up to my end of the agreement.

The gentleman——

Mr. HOWARD. Would the gentleman yield?

I would like to comment on this particular subject. Although I do not know of anyone who wishes to personally pursue Mrs. Burford further in this instance, when we talk about promise and performance concerning documents to which we will have access, I believe that there is involved in this a problem much more basic than Mrs. Burford.

That has to do with the performance of the Department of Justice and the U.S. attorney in response to the contempt citation that was voted overwhelmingly by the House.

I believe that the inaction over 3 months of the mandate by the Congress to the U.S. attorney is something that must be addressed, and addressed, very forthrightly, because I believe that there is a constitutional problem here that will affect the rights of the Congress for many years in the future.

I believe that another committee, the Committee on Judiciary, has begun hearings on that. I hope they will pursue them with great vigor.

I have notified the chairman of that committee that, should they not move forcefully and quickly on that, they believe that our committee here on Public Works and Transportation who were the ones who had to initiate this contempt citation, have every right to hold hearings ourselves.

I hope that is not necessary. So, as we say, without prejudice, I would feel, and have notified people representing Mrs. Burford, that this would be held in abeyance.

I do not believe that in the end there will be any difficult time for Mrs. Burford. But I believe that we do have to move forward to see that this mandate voted by this committee, voted by the Congress, certified by the Speaker to the U.S. attorney—we must have some concrete answers as to why it might be possible for a U.S. attorney to do absolutely nothing——

Mr. SNYDER. Would the chairman yield briefly?

Mr. HOWARD. I would be happy to—I believe the gentleman from New York has the time.

Mr. MOLINARI. I would like to answer that first, if you don't mind, Mr. Snyder.

I was as forceful as anybody in the House in seeking the contempt citation. I think I might have been the first Member of Congress that called for her resignation or dismissal.

The fact is that at this point in time she is not on the scene. She is gone. She has absolutely no control over any documents that are to be delivered to this subcommittee or any other committee.

So it appears to me that she occupies a very uncomfortable and you might say unfair position in that she is being held hostage while we wrestle with Justice Department.

Indeed, I think the Department of Justice deserves the major share of the blame for the impasse that occurred here. I think previous testimony before this subcommittee illustrated that, indeed, Mrs. Burford wanted to give those documents to us in the beginning.

She was prevented from doing so by the Department of Justice. She went through her own anguish and she resigned. I would like to have seen her resign sometime before so we could have gotten on with the job of restructuring EPA.

But she now sits back someplace awaiting action. In the spirit of fairness, I think Congress owes it to her to move ahead as quickly as possible.

I hope that is something we can all do in a bipartisan spirit.

Mr. HOWARD. If the gentleman will yield, I believe that we can. That depends on how quickly we move on this much more basic situation.

I don't believe we can wipe this out and still get to the point as well as we could if we handled this first. I think that it will be done rather quickly.

There were others who had called for Mrs. Burford's resignation. I believe I did in June 1981.

Mr. LEVITAS. The gentleman from Kentucky.

Mr. SNYDER. Thank you. I just want to reiterate what the gentleman from New York said. Mrs. Burford's predicament is as a result of what she was instructed to do by her boss as a result of the Justice Department's advice, as I understand it.

I think that is pretty clear for the record around here. The chairman's criticism of Justice is probably justified and I believe it is. But to hold her hostage, and to persecute her because of what Justice is wrongly doing or Justice is not doing I think is inherently improper.

If Justice is not doing their duty, they are the ones that ought to be sought out, and I trust that they will be and not this lady.

I talked to her once on the telephone during the whole time that she was at EPA. But it just strikes me that she wanted to comply with Congress' request.

She was told by the boss not to comply. She had the choice of either following the boss' instructions, or doing what Mr. Ruckleshaus did in years past in regard to Mr. Archibald Cox. Maybe his decision was proper and hers was wrong.

But I think that we have to understand why she did what she did and understand that it really is not her fault, but the fault of Justice.

Mr. HOWARD. Perhaps I did not make myself clear. I do not believe there was any intention of persecution or prosecution concerning Mrs. Burford.

I believe that she has been told that several times. That situation seems to prevail in the Congress and will prevail. I believe that there is a monumental question and problem that must be resolved.

I believe that that should come first. I feel Mrs. Burford should not feel she is being held hostage or in any personal danger of being put in prison.

Mr. SNYDER. But, Mr. Chairman, she is being held hostage to the Congress for not just her, but the Congress' belief that Justice has not acted properly in this case. I just think that is wrong.

Mr. HOWARD. I just do not want it conveyed that somehow, in a few days, without serious opposition, that there may be some effort to wipe this out. Perhaps then give people a chance to say that whether or not the U.S. attorney was proper in making his own decision, as he says, to just ignore the Congress of the United States, whether that might be moot.

I want to see that that does not happen.

Mr. HOWARD. I think the judgment——

Mr. SNYDER. The chairman of the subcommittee has made a commitment. If those documents that he made that commitment for are delivered, then we have an obligation to follow through.

Mr. **Howard**. I believe the chairman of the subcommittee has every right to pursue what he wishes. I just want to make it clear that as that moves from the subcommittee chairman toward perhaps the Speaker of the House, that there will be some opposition to it.

I just don't want it to be stated that that will be a fait accompli. The chairman of the subcommittee and I have discussed this many times.

I just hope this whole thing can be resolved very, very quickly. But I believe there are basics that must be followed here concerning the rights of the Congress.

I thank the gentleman for yielding.

Mr. **Levitas**. Does any other member of the subcommittee desire to make a statement at this point?

If not, let me just make an observation, if I may conclude this portion of the discussion, which I think is very important to get out. I have said and I repeat again, I intend to live up to the commitments undertaken and I agreed to with the administration relating to the access to these documents.

I am advised that if I should be satisfied that there has been compliance and implementation, that I will introduce a resolution which will deal with the matter. I don't want to get into the specifics of what it will do, because there is a substantial legal question whether you can purge a criminal contempt.

But I will introduce a resolution which I understand will then be referred to this subcommittee for its action, and in the event we favorably report it, it will be reported to the full committee.

If the full committee favorably reports it, it will be referred to the House. I understand that is the procedure that would be followed.

It is my intention that when there is compliance with the undertakings that have been entered into by the administration, I will do what I have agreed to do, and I will do so in total good faith.

Having said all of that—and I don't want to in any way let you think that what I am about to say derogates from that—back in December I suggested that the Judiciary Committee should examine the actions and lack of actions of the U.S. attorney and the Attorney General of the United States to ascertain whether they committed impeachable offenses by failing to faithfully execute the law and blatantly flaunting the statute under which the criminal contempt citation was brought.

I still feel that way. I am going to do everything I can do, either through the Judiciary Committee or, Mr. Chairman, through your committee, to find out how it is a U.S. attorney, or Attorney General can decide when they are going to obey the law particularly with regard to a criminal contempt citation by Congress.

Mr. **Snyder**. Will the gentleman yield?

I just want to say I have no disagreement with your statement.

Mr. **Levitas**. Thank you. Now, turning to the specific events of today, during last week's hearing on March 16, it was disclosed that on June 1, 1982, efforts by the U.S. Environmental Protection Agency region VI office in Dallas, TX, to have three lead smelting companies clean up areas of lead soil contamination in neighborhoods surrounding these companies were halted by the Environmental Protection Agency headquarter's decision, a decision that Dr. John Hernandez, the at-that-time EPA Deputy Administrator, advised this subcommittee he made.

As to why Dr. Hernandez advised that, and I quote:

> We did not have the kind of information as to exposure to particularly children, but individuals in that area as a whole.
>
> What we really needed was a sampling program where we went out and actually measured people, blood levels in children, particularly.

The question that we are going to explore today is what was occuring in Dallas, TX, in this matter, and whether or not the decision not to go forward with the cleanup, and to make decisions for removal of the potential health hazard to individuals and particularly children was a reflection of a change or new policy of this administration.

If that were the case, I think it would have very serious implications. It is for that reason that we have asked the witnesses today to participate in this further investigation.

I am very pleased that our first witness today is the Congressman who represents this area of Dallas, TX, a very good friend of mine, one of our most able and competent colleagues who I know has been very interested and concerned about this matter from its inception.

I am very pleased to welcome as our first witness today the Honorable Martin Frost, Member of Congress, from Texas.

TESTIMONY OF HON. MARTIN FROST, A REPRESENTATIVE IN CONGRESS FROM THE STATE OF TEXAS

Mr. Frost. Mr. Chairman, I appreciate the opportunity to appear before your subcommittee today and want to begin by expressing my gratitude on behalf of my constituents for your vigorous and persistent investigation into the actions of the Environmental Protection Agency in the lead contamination problem in Dallas.

The three lead smelters involved are located in the 24th Congressional District of Texas, which I represent, and the people who live near these plants are my constituents.

Let me also add that I have fought many battles with Federal bureaucrats during my service in Congress, but none has been more frustrating nor more protracted than trying to seek relief for the people of West Dallas and Oak Cliff who live in the shadow of these three smelters.

I would like to provide the subcommittee with a brief chronology of my involvement in this matter along with some observations about the manner in which the entire situation has been approached by the EPA.

I was first contacted about lead contamination in the summer of 1981 by a constituent who was a former employee of the smelter operated by the RSR Corp. He was concerned about the health of his children since he lived near the plant where he worked.

Earlier that summer, the first of three studies showing high concentrations of lead in the soil near the smelters was made public by the EPA raising the concerns of many residents of the affected areas.

I began a series of discussions with the regional office of the EPA on the Dallas problem and subsequently wrote to EPA on September 11, 1981, making a number of suggestions about further testing of residents of the area to determine the exact extent of lead contamination.

A copy of my letter to EPA is being submitted to this subcommittee along with my statement. I also sent a postal patron mailing to every household near the three plants informing them of the times and locations of blood testing that was to be conducted by the city of Dallas.

During the succeeding months, I held a series of meetings with Dr. John Hernandez, various other officials of the EPA and an attorney representing one of the smelters, in an attempt to seek a speedy resolution of the problem.

In reviewing my files, I have determined the following. On September 9, 1981, I was visited by an attorney representing RSR Corp. He confirmed earlier published reports that RSR had volunteered to the EPA that it would replace soil if they found lead levels in excess of 1,000 parts per million at any particular location.

He told me RSR had already removed lead-contaminated soil from the playground at the Navarro Elementary School in the West Dallas area near the RSR plant and that they were prepared to do the same at the Tabernacle Day Care Center, also located in West Dallas.

Further, he informed me that his company had volunteered to help develop protocols in soil-lead level testing for the EPA.

On October 14, 1981, I met with Dr. Hernandez in my office in Washington and urged him to move forward quickly with the analysis of the second lead study then being conducted by the city of Dallas so that EPA could initiate remedial action.

I raised the question of whether Superfund money could be used for any cleanup of the area and what options were under consideration. He told me that soil removal was one of the options under consideration and that it would take 2 or 3 months to analyze the results of the tests once they were completed.

He said the analysis could be ready by March and that remedial actions could be recommended by July 1, 1982.

On April 22, 1982, I met with Dr. Hernandez again in my office in Washington and he advised me that because the testing methodology used by the city of Dallas in the fall of 1981 was insufficiently scientific, a new round of tests would be required before EPA could initiate action.

He said that EPA was confused as to the source of the lead contamination and that they needed to retest to determine if blood lead levels could be found that were statistically significant or that those levels were directly correlated to high lead levels in the soil.

He stated that there was no national soil lead standard and that EPA would like to preclude setting one. Dr. Hernandez said that if EPA found high blood lead levels and a correlation with lead in the soil then they probably would undertake soil replacement.

He said they would, under those circumstances, negotiate cleanup costs with the companies and if EPA was unable to get the companies to pay for the costs, EPA would consider using Superfund money for this purpose.

Of course, the third and most recent blood lead and soil contamination study released by the EPA on February 1 of this year showed that a number of preschool children living near the RSR plant did have elevated blood lead levels.

In fact, the study showed that 5.6 percent of the children living within one-half mile of the plant were lead toxic and that 10.5 percent had elevated blood lead levels which are levels of concern according to Centers for Disease Control standards.

The study showed high concentrations of lead in the soil near the RSR plant. In fact, the study showed that the 10 percent most contaminated soil contained 1,016 parts per million and the 5 percent most contaminated soil contained 1,445 parts per million.

The study concluded that soil lead levels could not be ignored as a contributing factor to elevated blood lead levels.

The study also showed that RSR was not in compliance with national ambient air quality standards for lead during 1982 testing periods and also concluded that the soil lead contamination was clearly the result of the plant's emissions and not directly attributable to auto emissions.

In August 1982, I wrote to the Texas Air Control Board concerning the reported failure of RSR to comply with air standards and urged TACB to take immediate enforcement action. I contacted that agency again in February of this year after the release of the lead study confirmed the reports of the previous summer and, again, urged TACB to take forceful enforcement action.

Finally, some action has begun. On Tuesday, March 22 of this week, the executive director of that State agency recommended to the board that the State seek injunctive relief which may include financial penalties. For the subcommittee's information, I have also enclosed my letters to TACB along with my statement.

Notwithstanding all of this evidence, EPA has taken the position that the problem was not serious enough to merit EPA action and that the problem should be resolved by State and city agencies.

I wrote Dr. Hernandez protesting this conclusion on February 7. A copy of my letter to him and his response of March 9 are also attached to my statement.

I should like to point out to this subcommittee that prior to my meetings in October 1981 and April 1982 when Dr. Hernandez told me that soil removal by the smelters and use of Superfund money for cleanup were under active consideration, that he and other EPA officials in Washington had apparently advised the smelters not to actively pursue soil replacement and had apparently blocked consideration of using Superfund money for this purpose.

This is clear from previous testimony before your subcommittee and from published reports in the Texas media detailing information from internal EPA working papers obtained under Freedom of Information requests.

The people who live in the shadows of these three smelters are poor blacks and poor Mexican Americans. In most cases, they cannot afford to move away. They depend upon their Government through the EPA and the Congress to protect the health of their children.

If we do not stand up for their rights, no one will. They have every bit as much a right to be free from environmental hazards as do President Reagan's wealthiest friends in California or in the affluent sections of Dallas.

EPA has had ample time to determine that there is a problem in the areas surrounding these lead smelters in Dallas and to do something to remedy it. The owners of the smelters themselves were willing to help remedy the problem and would have done so already if they had not been dissuaded from proceeding by EPA officials at the highest level.

I urge this committee to continue its investigation into the lead problem in Dallas and not to stop until the truth has been laid bare. What has happened in Dallas could, most tragically, occur anywhere in the United States.

Mr. LEVITAS. Thank you, Congressman Frost. Without objection, your statement and the attachments thereto will be made part of the record.

[The statement of Mr. Frost follows:]

STATEMENT OF THE HONORABLE MARTIN FROST
BEFORE THE SUBCOMMITTEE ON INVESTIGATIONS AND OVERSIGHT
HOUSE COMMITTEE ON PUBLIC WORKS
THURSDAY, MARCH 24, 1983

Mr. Chairman, I appreciate the opportunity to appear before your Subcommittee today and want to begin by expressing my gratitude on behalf of my constituents for your vigorous and persistent investigation into the actions of the Environmental Protection Agency in the lead contamination problem in Dallas. The three lead smelters involved are located in the 24th Congressional District of Texas, which I represent, and the people who live near these plants are my constituents.

Let me also add that I have fought many battles with federal bureaucrats during my service in Congress, but none has been more frustrating nor more protracted than trying to seek relief for the people of West Dallas and Oak Cliff who live in the shadow of these three smelters.

I would like to provide the subcommittee with a brief chronology of my involvement in this matter along with some observations about the manner in which the entire situation has been approached by the EPA.

I was first contacted about lead contamination in the summer of 1981 by a constituent who was a former employee of the smelter operated by the RSR Corporation. He was concerned about the health of his children since he lived near the plant where he worked. Earlier that summer, the first of three studies showing high concentrations of lead in the soil near the smelters was made public by the EPA raising the concerns of many residents of the affected areas. I began a series of discussions with the regional office of the EPA on the Dallas problem and subsequently wrote to EPA on September 11, 1981 making a number of suggestions about further testing of residents of the area to determine the exact extent of lead contamination. A copy of my letter to EPA is being submitted to this Subcommittee along with my statement. I also sent a postal patron mailing to every household near the three plants informing them of the times and locations of blood testing that was to be conducted by the City of Dallas.

During the succeeding months, I held a series of meetings with Dr. John Hernandez, various other officials of the EPA and an attorney representing one of the smelters, in an attempt to seek a speedy resolution of the problem.

In reviewing my files, I have determined the following:

(1) On September 9, 1981, I was visited by an attorney representing RSR Corporation. He confirmed earlier published reports that RSR had volunteered to the EPA that it would replace soil if they found lead levels in excess of 1,000 parts per million at any particular location. He told me RSR had already removed lead-contaminated soil from the playground at the Navarro Elementary School in the West Dallas area near the RSR plant and that they were prepared to do the same at the Tabernacle Day Care Center, also located in West Dallas. Further, he informed me that his company had volunteered to help develop protocols in soil-lead level testing for the EPA.

(2) On October 14, 1981, I met with Dr. Hernandez in my office in Washington and urged him to move forward quickly with the analysis of the second lead study then being conducted by

the City of Dallas so that EPA could initiate remedial action. I raised the question of whether Superfund money could be used for any cleanup of the area and what options were under consideration. He told me that soil removal was one of the options under consideration and that it would take two or three months to analyze the results of the tests once they were completed. He said the analysis could be ready by March and that remedial actions could be recommended by July 1, 1982.

(3) On April 22, 1982, I met with Dr. Hernandez again in my office in Washington and he advised me that because the testing methodology used by the City of Dallas in the fall of 1981 was insufficiently scientific, a new round of tests would be required before EPA could initiate action. He said that EPA was confused as to the source of the lead contamination and that they needed to retest to determine if blood lead levels could be found that were statistically significant or that those levels were directly correlated to high lead levels in the soil. He stated that there was no national soil lead standard and that EPA would like to preclude setting one. Dr. Hernandez said that if EPA found high blood lead levels and a correlation with lead in the soil then they probably would undertake soil replacement. He said they would, under those circumstances, negotiate cleanup costs with the companies and if EPA was unable to get the companies to pay for the costs, EPA would consider using Superfund money for this purpose.

Of course, the third and most recent blood lead and soil contamination study released by the EPA on February 1 of this year showed that a number of pre-school children living near the RSR plant did have elevated blood lead levels. In fact, the study showed that 5.6 percent of the children living within one-half mile of the plant were lead toxic and that 10.5 percent had elevated blood lead levels which are levels of concern according to Centers for Disease Control standards.

The study showed high concentrations of lead in the soil near the RSR plant. In fact, the study showed that the 10 percent most contaminated soil contained 1016 parts per million and the five percent most contaminated soil contained 1445 parts per million. The study concluded that soil lead levels could not be ignored as a contributing factor to elevated blood lead levels.

The study also showed that RSR was not in compliance with national ambient air quality standards for lead during 1982 testing periods and also concluded that the soil lead contamination was clearly the result of the plant's emissions and not directly attributable to auto emissions. In August of 1982, I wrote to the Texas Air Control Board concerning the reported failure of RSR to comply with air standards and urged TACB to take immediate enforcement action. I contacted that agency again in February of this year after the release of the lead study confirmed the reports of the previous summer and again urged TACB to take forceful enforcement action. Finally some action has begun: on Tuesday, March 22, the executive director of that state agency recommended to the Board that the State seek injunctive relief which may include financial penalties. For the subcommittee's information, I have also enclosed my letters to TACB along with my statement.

Notwithstanding all of this evidence, EPA has taken the position that the problem was not serious enough to merit EPA action and that the problem should be resolved by State and City agencies. I wrote Dr. Hernandez protesting this conclusion on February 7. A copy of my letter to him and his response of March 9 are also attached to my statement.

I should like to point out to this subcommittee that prior to my meetings in October of 1981 and April of 1982 when Dr. Hernandez told me that soil removal by the smelters and use of Superfund money for cleanup were under active consideration, that he and other EPA officials in Washington had apparently advised the smelters not to actively pursue soil replacement and had apparently blocked consideration of using Superfund money for this purpose. This is clear from previous testimony before your subcommittee and from published reports in the Texas media detailing information from internal EPA working papers obtained under Freedom of Information requests.

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EPA has had ample time to determine that there is a problem in the areas surrounding these lead smelters in Dallas and to do something to remedy it. The owners of the smelters themselves were willing to help remedy the problem and would have done so already if they had not been dissuaded from proceeding by EPA officials at the highest level. I urge this committee to continue its investigation into the lead problem in Dallas and not to stop until the truth has been laid bare. What has happened in Dallas could, most tragically, occur anywhere in the United States.

MARTIN FROST
24TH DISTRICT, TEXAS

RULES COMMITTEE

MAJORITY WHIP AT LARGE

1238 LONGWORTH HOUSE OFFICE BUILDING
WASHINGTON, D.C. 20515
(202) 225-3605

Congress of the United States
House of Representatives
Washington, D.C. 20515

DISTRICT OFFICES:
DALLAS, TEXAS 75208
☐ OAK CLIFF BANK TOWER, ROOM 1319
400 SOUTH ZANG BOULEVARD
(214) 941-6252
GRAND PRAIRIE, TEXAS 75050
☐ 211 WEST MAIN, ROOM 109
(214) 262-1503
ARLINGTON, TEXAS 76010
☐ 103 SOUTH MESQUITE
(817) 261-7799
PLEASE REPLY TO
OFFICE CHECKED

September 11, 1981

Ms. Frances Phillips
Acting Regional Administrator
Environmental Protection Agency
1201 Elm St.
Dallas, TX 75270

Dear Ms. Phillips:

I have been and continue to be concerned about the possibility of lead contamination among citizens of Dallas who reside near the city's lead smelting plants. I am encouraged by the cooperation shown so far by the EPA, the City of Dallas, and the Centers for Disease Control in the effort to identify and solve this troubling problem. However, I would like to make some further recommendations which I believe could improve the overall understanding of the problem as well as ultimately lead to a more thorough resolution of the situation.

I understand that the City plans a two-step testing process to determine the extent of lead contamination in citizens in the affected areas of Dallas. The City will first conduct an erythrocyte protoporphyrin (EP) test for all possible affected residents to determine potential lead toxicity. Then, for those individuals whose EP tests indicate possible toxicity, the City plans to conduct a blood level test, with a reading of 30 micrograms per deciliter of lead in the blood being the indicator of lead contamination.

At this time, I am recommending a series of other steps which I believe will be more comprehensive than the two-step process proposed by the City:

1. I am recommending that the City conduct simultaneous EP and blood level testing for all residents of the affected areas, instead of the two-step process originally proposed by the City. I understand that the EPA has requested the City to conduct this type of simultaneous testing for a small sample of residents from the affected areas. However, I believe that simultaneous testing for all residents in the targeted areas would provide the most comprehensive data possible and would enable us to determine the true extent of the problem.

2. I am recommending that a computer correlation study be conducted to determine if there is any statistically-significant relationship between the lead testing and the academic achievement tests of school-age children in the affected areas. Such a testing -- while admittedly costly in terms of both physical and financial resources -- might be a good indicator of the potential long-range effects of the lead contamination on the educational performance of the affected children.

3. I am recommending that the owners of the lead smelting plants be encouraged -- if not required -- to contribute financial resources to help pay for the simultaneous EP and blood level testing and for the computer correlation study.

I believe these three recommendations, if implemented, would give the citizens of Dallas a more complete understanding of the potential lead-contamination problems that currently exist and that have occurred in the past. I am willing to do whatever I can as a Member of Congress to facilitate all of these procedures. I have already pledged to distribute a "postal patron" mailing to all households in the affected area to alert those residents to the testing program being conducted by the City of Dallas. And I am willing to commit all resources available to me to do whatever is necessary to help resolve this difficult problem.

I will appreciate your consideration of these recommendations and I look forward to working with you and your office in the next few weeks.

Sincerely,

Martin Frost

MF:tb

MARTIN FROST
24th District, Texas

RULES COMMITTEE

MAJORITY WHIP AT LARGE

1238 Longworth House Office Building
Washington, D.C. 20515
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400 South Zang Boulevard
(214) [illegible]
GRAND PRAIRIE, TEXAS 75050
☐ 211 West Main, Room 106
(214) [illegible]
ARLINGTON, TEXAS 76010
☐ 100 South Mesquite
(817) [illegible]
Please reply to
office checked

August 5, 1982

Mr. Eli Bell, Deputy Director
Control and Prevention Program
Texas Air Control Board
6330 Hwy. 290 East
Austin, Texas 78723

Dear Mr. Bell:

I am writing to let you know of my continuing deep concern about the lead contamination surrounding the RSR Corporation plant and the Dixie Metals plant in Dallas. It is my understanding the Texas Air Control Board is presently conducting air monitoring tests in the immediate vicinity of these plants and that preliminary data shows that national standards established by the Environmental Protection Agency are being exceeded in the vicinity of the RSR plant.

Mr. Cecil Bradford of your office has informed Ms. Kristi Walseth of my Washington office that TACB can expect to have more definitive information on these tests by the end of this month and at that time the test results will be turned over to you for further action. I would like to take this opportunity to encourage you to move expeditiously to take whatever enforcement action may be warranted should test results show that, in fact, RSR is not in compliance with the national lead standards. I am very concerned that RSR, and possibly Dixie Metals, may be continuing to emit dangerous levels of lead which may complicate an already volatile situation.

I would very much appreciate your keeping my office informed of your progress in this matter. Also, thank you for your prompt and courteous response to our initial inquiry.

I look forward to hearing from you in the very near future.

Sincerely,

Martin Frost

Martin Frost

MF/kew

cc: Mr. Cecil Bradford

MARTIN FROST
24th District, Texas

RULES COMMITTEE

MAJORITY WHIP AT LARGE

[illegible] Longworth House Office Building
Washington, D.C. 20515
[illegible]

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(214) [illegible]

ARLINGTON, TEXAS [illegible]
☐ 100 South Mesquite
(817) [illegible]

Please reply to
office checked

February 3, 1983

Mr. Bill Stewart, P.E.
Executive Director
Texas Air Control Board
6330 Hwy. 290 East
Austin, Texas 78723

Dear Mr. Stewart:

I am writing to comment on the Dallas Area Lead Assessment Study report released by the Environmental Protection Agency on February 1, 1983. I am particularly concerned about the results of the study which show the RSR plant exceeded national ambient air quality standards for lead in the testing period of April-July, 1982. The figures in the report show that Monitor A, located 50 yards from the plant registered 2.97ug/m3 and Monitor B, located 500 yards from the plant registered 1.57ug/m3. It is my understanding that the national ambient air quality standard for lead is 1.5ug/m3.

On August 5, 1982, I wrote to Mr. Eli Bell, Deputy Director of the Control and Prevention Program, to express my concern about this very same subject following an article published in the Fort Worth Star Telegram of August 1. That article reported the RSR plant was exceeding the national standards for lead by a factor of two during the April through June testing period.

In your response to my letter, dated August 12, you stated that the Texas Air Control Board would "initiate appropriate action to reduce ambient levels of lead which exceed the standard." Further, your letter stated that if it was determined that RSR emissions cause of contribute to any exceedances of the lead standard, TACB would seek "immediate voluntary emission reductions from the company," and if those voluntary reductions were not forthcoming, TACB would take additional steps including a formal Board Order or revision of the State Implementation Plan for lead which would require additional controls or court action.

The EPA study has now conclusively established that the elevated blood lead levels among preschool black children living in the vicinity of the RSR plant are the direct result of air emissions from the plant and not from traffic in the neighborhood.

Mr. Bill Stewart, P.E.
Page Two
February 3, 1983

Since the plant is in violation of the national lead standards and it has been shown to be the cause of the elevated blood lead levels of those children, I now expect strong and immediate action from TACB to resolve this situation.

I have asked State Senator Oscar H. Mauzy, whose State Senatorial District includes the area where the plant is located and the children live, to assist me in bringing about prompt action by the Board. Both Senator Mauzy and I will follow the progress of TACB in this matter with great interest and concern.

I would very much appreciate your keeping my office informed of your progress in this matter. I look forward to hearing from you in the very near future.

Sincerely,

Martin Frost

MF/kew

cc: Senator Oscar H. Mauzy
Mr. Dick Whittington

.MARTIN FROST
24th District, Texas

RULES COMMITTEE

MAJORITY WHIP AT LARGE

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Congress of the United States
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(214) [illegible]-1503
ARLINGTON, TEXAS 76010
☐ [illegible] South Mesquite
(817) [illegible]
Please reply to
office checked

February 7, 1983

John W. Hernandez, Ph.D.
Deputy Administrator
Environmental Protection Agency
401 M Street, S.W.
Washington, D.C. 20460

Dear Dr. Hernandez:

I have carefully reviewed the information released by the Environmental Protection Agency on February 1, 1983 relating to the Dallas Area Lead Assessment Study. The results of that study are of continuing concern to my constituents and to myself.

Specifically, it is my understanding from reviewing the materials released by EPA that 10.5 percent of the black preschool children living within one-half mile of the RSR plant have blood lead levels of 30 ug/dl or greater. Of those children, 5.6 percent have been classified as having lead toxicity, which is defined as a blood lead level of 30 ug/dl or greater and an FEP level of 50 ug/dl or greater.

According to results of a study conducted by the National Center for Health Statistics between 1976-1980, the national average for black preschool children with blood lead levels of 30 ug/dl or greater is 12.2 percent of the surveyed population. According to that study, "30 ug/dl is the cut-off used in the CDC Lead Screening Program for referring children for followup." It is my understanding from discussions with EPA and CDC that no national standard exists for lead toxicity, or the combination of blood lead levels and FEP. As such, there is no way to compare the 5.6 percent of the affected preschool children with lead toxicity in Dallas to national averages for lead toxicity.

However, I would like to take this opportunity to express my grave concern that the Dallas figures, when expressed in terms of blood lead levels only, are perilously close to the national average. Yet, in my discussions with your agency, the national average of 12.2 percent has been consistently compared with the 5.6 percent of the Dallas children with lead toxicity. It seems to me that this is comparing oranges with apples when the 12.2 percent is based only on blood lead levels and the 5.6 percent is based on blood lead and FEP.

Since the percentage of children in the surveyed Dallas population is so close to the national average, I believe this should require additional consideration by the EPA as to what remedial action could be taken by the agency, rather than to simply drop the ball in the lap of the City of Dallas.

I should also point out that the results of the air monitoring conducted by the Texas Air Control Board show that the RSR plant was in violation of the national ambient air quality standards for lead in at least two testing quarters during 1982.

I do not want to see this matter dismissed simply because a study has been completed and the number of affected children does not meet some arbitrary level established by EPA as what would require further action. The fact that 5.6 percent of the children are now officially classified as having lead toxicity and that 10.5 percent have blood lead levels which concern the CDC concerns the residents of the area, and as their elected representative, greatly concerns me.

As such, I should like to take this opportunity to ask exactly what remedial action the EPA can and will take in this situation and if the agency is not going to take any remedial action, I should like to know why.

I look forward to hearing from you in the very near future.

Sincerely,

Martin Frost

MF/kew

cc: Mr. Dick Whittington
Administrator, Region VI

MAR 11 1983

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

MAR 9 1983

OFFICE OF
THE ADMINISTRATOR

Honorable Martin Frost
House of Representatives
Washington, D.C. 20515

Dear Mr. Frost:

Thank you for your letter of February 7, 1983, regarding the Dallas Lead Study in which you express concern about the results of the study. The study shows that 10.2 percent of the black preschool children living within one-half mile of the RSR facility have blood lead levels in excess of 30 ug/dl, and that 5.6 percent have lead toxicity; i.e., blood levels above 30 ug/dl and free erythrocyte protoporphyrin (FEP) level of 50 ug/dl or greater.

There seems to be some confusion about the comparison of elevated blood levels and lead toxicity. Perhaps in your discussions with EPA, the data interpretations were not made sufficiently clear regarding the 5.6 percent lead toxicity value and the national figure of 12.2 percent elevated blood lead. As you observed, there is no national figure for lead toxicity contained in the lead study conducted between 1976-1980 by the National Center for Health Statistics for comparison with the 5.6 percent lead toxicity found in the Dallas lead study. You correctly asserted that the national figure of 12.2 percent for black preschool children with blood lead levels of 30 ug/dl or greater should be compared with the 10.2 percent with blood lead of 30 ug/dl or greater observed in the Dallas study.

Please be assured that EPA and the Centers for Disease Control (CDC) have consistently compared elevated blood lead levels in the Dallas study with the national figure of 12.2 percent elevated blood lead found in the National Center for Health Statistics Report. No comparison has been made between the 5.6 percent lead toxicity and the 12.2 percent figure in the national study; that is, apples with oranges. At the press conference, in statements made by CDC and EPA, and in the CDC and EPA lead study reports, we consistently compared the 10.2 percent blood lead levels with the national figure of 12.2 percent.

The quote you referred to from the national study that "30 ug/dl is the cut-off for referring children for followup" requires some clarification to be used in its proper context. According to the CDC guidelines (April 1978, page 5, Preventing Lead Poisoning in Young Children), there are four risk classifications for asymptomatic children, depending on a combination of the blood lead level and the FEP level. Children with a blood lead level of 30 ug/dl or above, but with an FEP level below 50 ug/dl, are placed in Class Ib. Class Ib is composed of children who appear to have transient, stable, or declining blood lead levels and are at a low risk for lead poisoning. In such a case, CDC recommends that the trend of exposure should be determined by repeat testing of these children. In the CDC report, recommendation No. 2 states that all children living near the sites and all children of workers at the smelters should be screened at 6-month intervals. In response to this recommendation, the city of Dallas has indicated that the city will initiate the suggested screening programs. Obviously, there is concern for any child with elevated blood lead levels, particularly above 30 ug/dl. At the press conference, we expressed our concerns about elevated blood lead levels in children. Recommendation No. 1 in the CDC report clearly states the medical concerns and specifically recommends steps to reduce the exposure of the children residing in the two areas. To this end, the city of Dallas and EPA have met with the lead companies and have requested that the smelters develop proposals to minimize lead exposure due to soil. We are awaiting their reply.

Concerning the ambient lead monitoring data that the Texas Air Control Board (TACB) has been collecting in the areas around Dixie Metals and RSR, we received the second quarter of quality assured monitoring data on January 31, 1983, and we agree it indicates a continuing problem that must be addressed. On February 11, 1983, we received the third quarter of quality assured monitoring data. Although the values around RSR are below the National Ambient Air Quality standard (i.e., 1.43 ug/m3) and are encouraging, we would not be satisfied that the area is in compliance with the standard until there is at least a full year of data. We are working with the TACB on continued data collection and in developing enforceable measures to reduce lead emissions from the RSR smelter. It is my understanding that the TACB and the city of Dallas met with RSR officials on Friday, February 18, in Austin. The meeting was an enforcement conference to review RSR's proposal to reduce lead emissions from their Dallas facility.

EPA is, and will be, closely following the State proceedings. In the unlikely case that the State and the city are not successful in their enforcement actions for RSR, EPA has authority under the Federal Clean Air Act to develop a Federal State Implementation Plan for the area. However, from our conversations with the State and the city, we do not anticipate this being necessary.

In summary, we are monitoring the TACB and city of Dallas air enforcement action with great interest and are awaiting the company's response to reduce the children's lead exposure in the neighborhood. I wish to point out that we are in no way simply dropping the ball in the lap of the city of Dallas, but are in fact working very closely with the mayor and his staff in a joint, coordinated, and cooperative manner to obtain an environmentally acceptable solution.

If you have further questions, or if I can assist you in any way, please call me.

Sincerely,

John W. Hernandez, Jr.
Deputy Administrator

A Special Issue Report from CONGRESSMAN MARTIN FROST

1238 LONGWORTH BUILDING
WASHINGTON, D.C. 20515
(202) 225-3605

LEAD CONTAMINATION TESTING PROGRAM

Dear Friends:

The City of Dallas is currently conducting tests for lead poisoning in pre-school and elementary age children. This action was made necessary by the fact that a study by the Environmental Protection Agency showed a high level of lead concentrated in the environment surrounding three lead smelters in Oak Cliff and West Dallas.

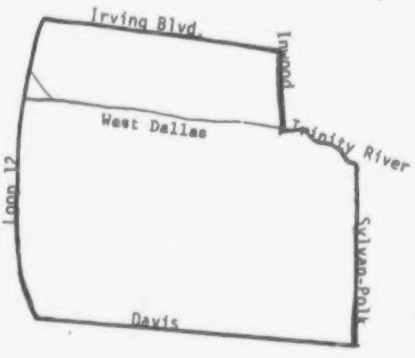
Lead poisoning is particularly dangerous to youngsters; however, once the current round of testing is completed (beginning on September 21 and continuing through October 17), tests will be given to adults who live within a two mile radius of the lead smelters. The City is particularly interested in testing very young children (ages 1-6).

The tests for lead poisoning require parents to give their consent, as a sample of the child's blood must be taken for examination. The tests are free to the participating individuals, and I would urge you to take advantage of this program for your family.

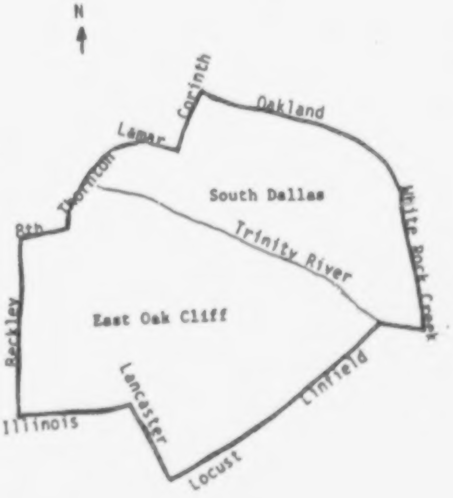
If you have a child attending elementary school and you live within the areas described by the maps shown below, you will receive a parental consent form to sign which will authorize the tests to be given. A complete list of the testing locations and times is shown on the reverse side of this special report.

The lead smelters are located at 2823 North Westmoreland, 1000 Sargent Road, and 3030 McGowan. The maps shown below outline those parts of Oak Cliff and West Dallas that are within a two mile radius of these smelters.

If you live within these areas and need more information, please give the City Health Department a call. Their telephone numbers are: 670-8775 or 670-6082. City personnel will be able to provide the locations and times for the testing of adults (now scheduled for October 19-23).



Irving Blvd.
Inwood
West Dallas
Trinity River
Loop 12
Sylvan-Polk
Davis



N
Corinth
Oakland
Lamar
Thornton
South Dallas
8th
Trinity River
White Rock Creek
East Oak Cliff
Beckley
Lancaster
Linfield
Illinois
Locust

TESTING AREAS

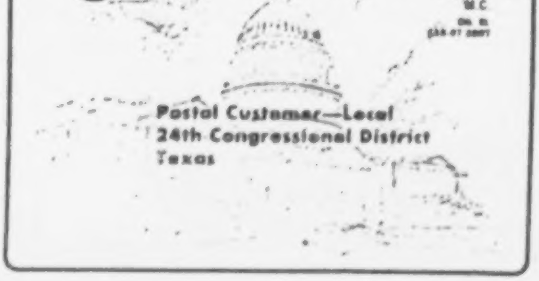
1981

BLOOD LEAD SCREENING PROGRAM SCHEDULE

September 21	Martin Luther King, Jr. Medical Building 2922 Martin Luther King Boulevard (9 a.m. - 3 p.m.)
September 22	Stevens Park School, 2615 Colorado (9 a.m. - 3 p.m.)
September 23	Charles Rice School, 2425 Pine Street (9 a.m. - 3 p.m.)
September 24	Sidney Lanier School, 1400 Walmsley (9 a.m. - 3 p.m.)
September 25	H. S. Thompson School, 5700 Bexar (9 a.m. - 3 p.m.)
September 26 (Sat.)	West Dallas Health Center, 3710 N. Hampton (9 a.m. - 3 p.m.)
September 28	Lorenzo Dezavala School, 3223 Conroe (9 a.m. - 3 p.m.)
September 29	William Brown Miller School, 3111 Bonnie View (9 a.m. - 3 p.m.)
September 30	Roger Q. Mills School, 1515 Lynn Haven (9 a.m. - 3 p.m.)
October 1	Jose Navarro School, 3530 Kingsbridge (9 a.m. - 3 p.m.)
October 2	Albert Sidney Johnston School, 2020 Mouser (9 a.m. - 3 p.m.)
October 3 (Sat.)	Oak Cliff Health Center, 300 North Ewing (9 a.m. - 3 p.m.)
October 5	Martin Luther King, Jr. Medical Building 2922 Martin Luther King Boulevard (9 a.m. - 3 p.m.)
October 6	N. W. Harllee School, 1216 East 8th (9 a.m. - 3 p.m.)
October 7	Priscilla Tyler School, 2333 Calypso (9 a.m. - 3 p.m.)
October 8	Gabe P. Allen School, 5220 Nomas (9 a.m. - 3 p.m.)
October 9	Colonial Elementary School, 1824 Pennsylvania (9 a.m. - 3 p.m.)
October 10 (Sat.)	West Dallas Health Center, 3710 N. Hampton (9 a.m. - 3 p.m.)
October 12	C. F. Carr School, 1952 Bayside (9 a.m. - 3 p.m.)
October 13	Amelia Earhart School, 3531 N. Westmoreland (9 a.m. - 3 p.m.)
October 14	George Washington Carver School, 3719 Greenleaf (9 a.m. - 3 p.m.)
October 15	Martin Luther King, Jr. Medical Building 2922 Martin Luther King Boulevard (9 a.m. - 3 p.m.)
October 16	West Dallas Health Center, 3710 N. Hampton (by appointment)
October 17 (Sat.)	Oak Cliff Health Center, 300 North Ewing (9 a.m. - 3 p.m.)

A SPECIAL REPORT

LEAD CONTAMINATION TESTING PROGRAM



Martin Frost
M.C.

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MARTIN FROST
24TH DISTRICT, TEXAS

RULES COMMITTEE

MAJORITY WHIP AT LARGE

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Congreſs of the United States
House of Representatives
Washington, D.C. 20515

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400 SOUTH ZANG BOULEVARD
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September 11, 1981

Ms. Frances Phillips
Acting Regional Administrator
Environmental Protection Agency
1201 Elm St.
Dallas, TX 75270

Dear Ms. Phillips:

I have been and continue to be concerned about the possibility of lead contamination among citizens of Dallas who reside near the city's lead smelting plants. I am encouraged by the cooperation shown so far by the EPA, the City of Dallas, and the Centers for Disease Control in the effort to identify and solve this troubling problem. However, I would like to make some further recommendations which I believe could improve the overall understanding of the problem as well as ultimately lead to a more thorough resolution of the situation.

I understand that the City plans a two-step testing process to determine the extent of lead contamination in citizens in the affected areas of Dallas. The City will first conduct an erythrocyte protoporphyrin (EP) test for all possible affected residents to determine potential lead toxicity. Then, for those individuals whose EP tests indicate possible toxicity, the City plans to conduct a blood level test, with a reading of 30 micrograms per deciliter of lead in the blood being the indicator of lead contamination.

At this time, I am recommending a series of other steps which I believe will be more comprehensive than the two-step process proposed by the City:

1. I am recommending that the City conduct simultaneous EP and blood level testing for all residents of the affected areas, instead of the two-step process originally proposed by the City. I understand that the EPA has requested the City to conduct this type of simultaneous testing for a small sample of residents from the affected areas. However, I believe that simultaneous testing for all residents in the targeted areas would provide the most comprehensive data possible and would enable us to determine the true extent of the problem.

2. I am recommending that a computer correlation study be conducted to determine if there is any statistically-significant relationship between the lead testing and the academic achievement tests of school-age children in the affected areas. Such a testing -- while admittedly costly in terms of both physical and financial resources -- might be a good indicator of the potential long-range effects of the lead contamination on the educational performance of the affected children.

3. I am recommending that the owners of the lead smelting plants be encouraged -- if not required -- to contribute financial resources to help pay for the simultaneous EP and blood level testing and for the computer correlation study.

I believe these three recommendations, if implemented, would give the citizens of Dallas a more complete understanding of the potential lead-contamination problems that currently exist and that have occurred in the past. I am willing to do whatever I can as a Member of Congress to facilitate all of these procedures. I have already pledged to distribute a "postal patron" mailing to all households in the affected area to alert those residents to the testing program being conducted by the City of Dallas. And I am willing to commit all resources available to me to do whatever is necessary to help resolve this difficult problem.

I will appreciate your consideration of these recommendations and I look forward to working with you and your office in the next few weeks.

Sincerely,

Martin Frost

Martin Frost

MF:tb

MARTIN FROST
24TH DISTRICT, TEXAS

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August 5, 1982

Mr. Eli Bell, Deputy Director
Control and Prevention Program
Texas Air Control Board
6330 Hwy. 290 East
Austin, Texas 78723

Dear Mr. Bell:

I am writing to let you know of my continuing deep concern about the lead contamination surrounding the RSR Corporation plant and the Dixie Metals plant in Dallas. It is my understanding the Texas Air Control Board is presently conducting air monitoring tests in the immediate vicinity of these plants and that preliminary data shows that national standards established by the Environmental Protection Agency are being exceeded in the vicinity of the RSR plant.

Mr. Cecil Bradford of your office has informed Ms. Kristi Walseth of my Washington office that TACB can expect to have more definitive information on these tests by the end of this month and at that time the test results will be turned over to you for further action. I would like to take this opportunity to encourage you to move expeditiously to take whatever enforcement action may be warranted should test results show that, in fact, RSR is not in compliance with the national lead standards. I am very concerned that RSR, and possibly Dixie Metals, may be continuing to emit dangerous levels of lead which may complicate an already volatile situation.

I would very much appreciate your keeping my office informed of your progress in this matter. Also, thank you for your prompt and courteous response to our initial inquiry.

I look forward to hearing from you in the very near future.

Sincerely,

Martin Frost

MF/kew

cc: Mr. Cecil Bradford

MARTIN FROST
24TH DISTRICT, TEXAS

RULES COMMITTEE

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February 3, 1983

Mr. Bill Stewart, P.E.
Executive Director
Texas Air Control Board
6330 Hwy. 290 East
Austin, Texas 78723

Dear Mr. Stewart:

I am writing to comment on the Dallas Area Lead Assessment Study report released by the Environmental Protection Agency on February 1, 1983. I am particularly concerned about the results of the study which show the RSR plant exceeded national ambient air quality standards for lead in the testing period of April-July, 1982. The figures in the report show that Monitor A, located 50 yards from the plant registered 2.97ug/m3 and Monitor B, located 500 yards from the plant registered 1.57ug/m3. It is my understanding that the national ambient air quality standard for lead is 1.5ug/m3.

On August 5, 1982, I wrote to Mr. Eli Bell, Deputy Director of the Control and Prevention Program, to express my concern about this very same subject following an article published in the Fort Worth Star Telegram of August 1. That article reported the RSR plant was exceeding the national standards for lead by a factor of two during the April through June testing period.

In your response to my letter, dated August 12, you stated that the Texas Air Control Board would "initiate appropriate action to reduce ambient levels of lead which exceed the standard." Further, your letter stated that if it was determined that RSR emissions cause of contribute to any exceedances of the lead standard, TACB would seek "immediate voluntary emission reductions from the company," and if those voluntary reductions were not forthcoming, TACB would take additional steps including a formal Board Order or revision of the State Implementation Plan for lead which would require additional controls or court action.

The EPA study has now conclusively established that the elevated blood lead levels among preschool black children living in the vicinity of the RSR plant are the direct result of air emissions from the plant and not from traffic in the neighborhood.

Since the plant is in violation of the national lead standards and it has been shown to be the cause of the elevated blood lead levels of those children, I now expect strong and immediate action from TACB to resolve this situation.

I have asked State Senator Oscar H. Mauzy, whose State Senatorial District includes the area where the plant is located and the children live, to assist me in bringing about prompt action by the Board. Both Senator Mauzy and I will follow the progress of TACB in this matter with great interest and concern.

I would very much appreciate your keeping my office informed of your progress in this matter. I look forward to hearing from you in the very near future.

Sincerely,

Martin Frost

MF/kew

cc: Senator Oscar H. Mauzy
Mr. Dick Whittington

.MARTIN FROST
24TH DISTRICT, TEXAS

RULES COMMITTEE

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(214) 262-1503

ARLINGTON, TEXAS 76010
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February 7, 1983

John W. Hernandez, Ph.D.
Deputy Administrator
Environmental Protection Agency
401 M Street, S.W.
Washington, D.C. 20460

Dear Dr. Hernandez:

I have carefully reviewed the information released by the Environmental Protection Agency on February 1, 1983 relating to the Dallas Area Lead Assessment Study. The results of that study are of continuing concern to my constituents and to myself.

Specifically, it is my understanding from reviewing the materials released by EPA that 10.5 percent of the black preschool children living within one-half mile of the RSR plant have blood lead levels of 30 ug/dl or greater. Of those children, 5.6 percent have been classified as having lead toxicity, which is defined as a blood lead level of 30 ug/dl or greater and an FEP level of 50 ug/dl or greater.

According to results of a study conducted by the National Center for Health Statistics between 1976-1980, the national average for black preschool children with blood lead levels of 30 ug/dl or greater is 12.2 percent of the surveyed population. According to that study, "30 ug/dl is the cut-off used in the CDC Lead Screening Program for referring children for followup." It is my understanding from discussions with EPA and CDC that no national standard exists for lead toxicity, or the combination of blood lead levels and FEP. As such, there is no way to compare the 5.6 percent of the affected preschool children with lead toxicity in Dallas to national averages for lead toxicity.

However, I would like to take this opportunity to express my grave concern that the Dallas figures, when expressed in terms of blood lead levels only, are perilously close to the national average. Yet, in my discussions with your agency, the national average of 12.2 percent has been consistently compared with the 5.6 percent of the Dallas children with lead toxicity. It seems to me that this is comparing oranges with apples when the 12.2 percent is based only on blood lead levels and the 5.6 percent is based on blood lead and FEP.

Since the percentage of children in the surveyed Dallas population is so close to the national average, I believe this should require additional consideration by the EPA as to what remedial action could be taken by the agency, rather than to simply drop the ball in the lap of the City of Dallas.

I should also point out that the results of the air monitoring conducted by the Texas Air Control Board show that the RSR plant was in violation of the national ambient air quality standards for lead in at least two testing quarters during 1982.

I do not want to see this matter dismissed simply because a study has been completed and the number of affected children does not meet some arbitrary level established by EPA as what would require further action. The fact that 5.6 percent of the children are now officially classified as having lead toxicity and that 10.5 percent have blood lead levels which concern the CDC concerns the residents of the area, and as their elected representative, greatly concerns me.

As such, I should like to take this opportunity to ask exactly what remedial action the EPA can and will take in this situation and if the agency is not going to take any remedial action, I should like to know why.

I look forward to hearing from you in the very near future.

Sincerely,

Martin Frost

MF/kew

cc: Mr. Dick Whittington
Administrator, Region VI

MAR 11 1983

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

MAR 9 1983

OFFICE OF
THE ADMINISTRATOR

Honorable Martin Frost
House of Representatives
Washington, D.C. 20515

Dear Mr. Frost:

Thank you for your letter of February 7, 1983, regarding the Dallas Lead Study in which you express concern about the results of the study. The study shows that 10.2 percent of the black preschool children living within one-half mile of the RSR facility have blood lead levels in excess of 30 ug/dl, and that 5.6 percent have lead toxicity; i.e., blood levels above 30 ug/dl and free erythrocyte protoporphyrin (FEP) level of 50 ug/dl or greater.

There seems to be some confusion about the comparison of elevated blood levels and lead toxicity. Perhaps in your discussions with EPA, the data interpretations were not made sufficiently clear regarding the 5.6 percent lead toxicity value and the national figure of 12.2 percent elevated blood lead. As you observed, there is no national figure for lead toxicity contained in the lead study conducted between 1976-1980 by the National Center for Health Statistics for comparison with the 5.6 percent lead toxicity found in the Dallas lead study. You correctly asserted that the national figure of 12.2 percent for black preschool children with blood lead levels of 30 ug/dl or greater should be compared with the 10.2 percent with blood lead of 30 ug/dl or greater observed in the Dallas study.

Please be assured that EPA and the Centers for Disease Control (CDC) have consistently compared elevated blood lead levels in the Dallas study with the national figure of 12.2 percent elevated blood lead found in the National Center for Health Statistics Report. No comparison has been made between the 5.6 percent lead toxicity and the 12.2 percent figure in the national study; that is, apples with oranges. At the press conference, in statements made by CDC and EPA, and in the CDC and EPA lead study reports, we consistently compared the 10.2 percent blood lead levels with the national figure of 12.2 percent.

The quote you referred to from the national study that "30 ug/dl is the cut-off for referring children for followup" requires some clarification to be used in its proper context. According to the CDC guidelines (April 1978, page 5, Preventing Lead Poisoning in Young Children), there are four risk classifications for asymptomatic children, depending on a combination of the blood lead level and the FEP level. Children with a blood lead level of 30 ug/dl or above, but with an FEP level below 50 ug/dl, are placed in Class Ib. Class Ib is composed of children who appear to have transient, stable, or declining blood lead levels and are at a low risk for lead poisoning. In such a case, CDC recommends that the trend of exposure should be determined by repeat testing of these children. In the CDC report, recommendation No. 2 states that all children living near the sites and all children of workers at the smelters should be screened at 6-month intervals. In response to this recommendation, the city of Dallas has indicated that the city will initiate the suggested screening programs. Obviously, there is concern for any child with elevated blood lead levels, particularly above 30 ug/dl. At the press conference, we expressed our concerns about elevated blood lead levels in children. Recommendation No. 1 in the CDC report clearly states the medical concerns and specifically recommends steps to reduce the exposure of the children residing in the two areas. To this end, the city of Dallas and EPA have met with the lead companies and have requested that the smelters develop proposals to minimize lead exposure due to soil. We are awaiting their reply.

Concerning the ambient lead monitoring data that the Texas Air Control Board (TACB) has been collecting in the areas around Dixie Metals and RSR, we received the second quarter of quality assured monitoring data on January 31, 1983, and we agree it indicates a continuing problem that must be addressed. On February 11, 1983, we received the third quarter of quality assured monitoring data. Although the values around RSR are below the National Ambient Air Quality standard (i.e., 1.43 ug/m3) and are encouraging, we would not be satisfied that the area is in compliance with the standard until there is at least a full year of data. We are working with the TACB on continued data collection and in developing enforceable measures to reduce lead emissions from the RSR smelter. It is my understanding that the TACB and the city of Dallas met with RSR officials on Friday, February 18, in Austin. The meeting was an enforcement conference to review RSR's proposal to reduce lead emissions from their Dallas facility.

EPA is, and will be, closely following the State proceedings. In the unlikely case that the State and the city are not successful in their enforcement actions for RSR, EPA has authority under the Federal Clean Air Act to develop a Federal State Implementation Plan for the area. However, from our conversations with the State and the city, we do not anticipate this being necessary.

In summary, we are monitoring the TACB and city of Dallas air enforcement action with great interest and are awaiting the company's response to reduce the children's lead exposure in the neighborhood. I wish to point out that we are in no way simply dropping the ball in the lap of the city of Dallas, but are in fact working very closely with the mayor and his staff in a joint, coordinated, and cooperative manner to obtain an environmentally acceptable solution.

If you have further questions, or if I can assist you in any way, please call me.

Sincerely,

John W. Hernandez

John W. Hernandez, Jr.
Deputy Administrator

A Special Issue Report from CONGRESSMAN MARTIN FROST

1238 LONGWORTH BUILDING
WASHINGTON, D.C. 20515
(202) 225-3605

LEAD CONTAMINATION TESTING PROGRAM

Dear Friends:

The City of Dallas is currently conducting tests for lead poisoning in pre-school and elementary age children. This action was made necessary by the fact that a study by the Environmental Protection Agency showed a high level of lead concentrated in the environment surrounding three lead smelters in Oak Cliff and West Dallas.

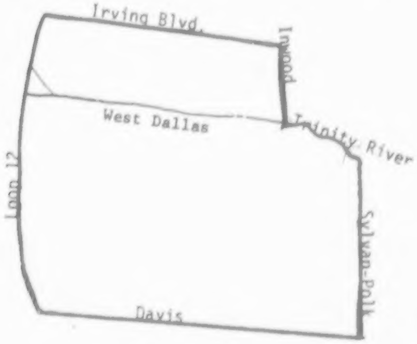
Lead poisoning is particularly dangerous to youngsters; however, once the current round of testing is completed (beginning on September 21 and continuing through October 17), tests will be given to adults who live within a two mile radius of the lead smelters. The City is particularly interested in testing very young children (ages 1-6).

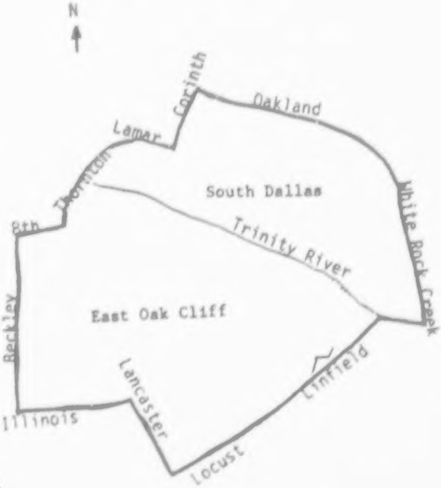
The tests for lead poisoning require parents to give their consent, as a sample of the child's blood must be taken for examination. The tests are free to the participating individuals, and I would urge you to take advantage of this program for your family.

If you have a child attending elementary school and you live within the areas described by the maps shown below, you will receive a parental consent form to sign which will authorize the tests to be given. A complete list of the testing locations and times is shown on the reverse side of this special report.

The lead smelters are located at 2823 North Westmoreland, 1000 Sargent Road, and 3030 McGowan. The maps shown below outline those parts of Oak Cliff and West Dallas that are within a two mile radius of these smelters.

If you live within these areas and need more information, please give the City Health Department a call. Their telephone numbers are: 670-8775 or 670-6082. City personnel will be able to provide the locations and times for the testing of adults (now scheduled for October 19-23).


Irving Blvd.
Inwood
West Dallas
Trinity River
Loop 12
Sylvan-Polk
Davis


N
Corinth
Oakland
Lamar
Thornton
South Dallas
8th
Trinity River
White Rock Creek
East Oak Cliff
Beckley
Lancaster
Linfield
Illinois
Locust

TESTING AREAS

1981

BLOOD LEAD SCREENING PROGRAM SCHEDULE

September 21	Martin Luther King, Jr. Medical Building 2922 Martin Luther King Boulevard (9 a.m. - 3 p.m.)
September 22	Stevens Park School, 2615 Colorado (9 a.m. - 3 p.m.)
September 23	Charles Rice School, 2425 Pine Street (9 a.m. - 3 p.m.)
September 24	Sidney Lanier School, 1400 Walmsley (9 a.m. - 3 p.m.)
September 25	H. S. Thompson School, 5700 Bexar (9 a.m. - 3 p.m.)
September 26 (Sat.)	West Dallas Health Center, 3710 N. Hampton (9 a.m. - 3 p.m.)
September 28	Lorenzo Dezavala School, 3223 Conroe (9 a.m. - 3 p.m.)
September 29	William Brown Miller School, 3111 Bonnie View (9 a.m. - 3 p.m.)
September 30	Roger Q. Mills School, 1515 Lynn Haven (9 a.m. - 3 p.m.)
October 1	Jose Navarro School, 3530 Kingsbridge (9 a.m. - 3 p.m.)
October 2	Albert Sidney Johnston School, 2020 Mouser (9 a.m. - 3 p.m.)
October 3 (Sat.)	Oak Cliff Health Center, 300 North Ewing (9 a.m. - 3 p.m.)
October 5	Martin Luther King, Jr. Medical Building 2922 Martin Luther King Boulevard (9 a.m. - 3 p.m.)
October 6	N. W. Harllee School, 1216 East 8th (9 a.m. - 3 p.m.)
October 7	Priscilla Tyler School, 2333 Calypso (9 a.m. - 3 p.m.)
October 8	Gabe P. Allen School, 5220 Nomas (9 a.m. - 3 p.m.)
October 9	Colonial Elementary School, 1824 Pennsylvania (9 a.m. - 3 p.m.)
October 10 (Sat.)	West Dallas Health Center, 3710 N. Hampton (9 a.m. - 3 p.m.)
October 12	C. F. Carr School, 1952 Bayside (9 a.m. - 3 p.m.)
October 13	Amelia Earhart School, 3531 N. Westmoreland (9 a.m. - 3 p.m.)
October 14	George Washington Carver School, 3719 Greenleaf (9 a.m. - 3 p.m.)
October 15	Martin Luther King, Jr. Medical Building 2922 Martin Luther King Boulevard (9 a.m. - 3 p.m.)
October 16	West Dallas Health Center, 3710 N. Hampton (by appointment)
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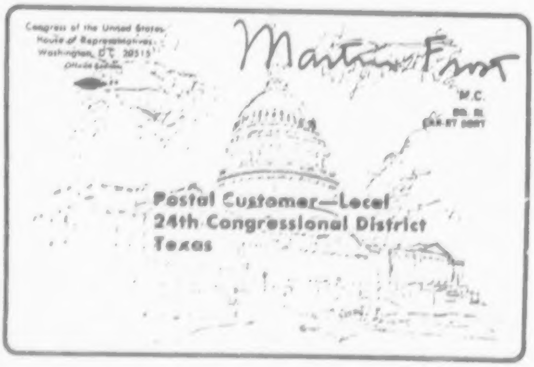
A SPECIAL REPORT

LEAD CONTAMINATION TESTING PROGRAM



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Mr. LEVITAS. I appreciate your testimony very much. It is quite informative. Your concern about this problem obviously has gone back some time. The only question I have about your testimony is that in the two meetings that you had with Dr. Hernandez, the one in 1981, and the second one in April 1982, I take it that you raised the question of availability of Superfund Program coverage of this matter on both of those occasions.

But I am not clear from your statement what Dr. Hernandez' response was. Did he say, yes, they are eligible? No, they are not; yes, they are eligible, we will do it? Yes, they are eligible; no, we won't do it? Or did you——

Mr. FROST. I have reviewed the notes taken by my staff from both of those meetings. As best I can determine from those notes, the response was that this is potentially the type of situation that could be covered by Superfund, and it was under consideration by his agency, but that they had not made any determination whether they would, in fact, attempt to cover this by Superfund moneys, but that it was at least potentially the type thing Superfund moneys could be applied to; that was an option.

Mr. LEVITAS. So far as you are aware, or so far as I am aware, that has not yet been designated as a Superfund site?

Mr. FROST. No; in fact, one of the original sites to receive Superfund moneys is in my district, in another part of my district, in Grand Prairie.

I believe that there are only a total of four sites in the entire State of Texas. Clearly, this is not one of the sites that has been put on any of their lists.

Mr. LEVITAS. Thank you, Congressman Frost. I am grateful for your participation.

Congressman Molinari?

Mr. MOLINARI. Just one question, Mr. Frost. You made reference to the fact that there were four sites in Texas on the priority list. Has the State of Texas evaluated this? Do they give a numerical index to the problem, like they do in my State, for example, in New York?

Mr. FROST. I really do not recall. I do know that the State of Texas has been involved, and in fact that the four sites were placed on the initial list at the recommendation of the State of Texas.

I do not recall in terms of numerical ranking how this was done.

Mr. MOLINARI. Do you have any knowledge as to the State's opinion as to the health problem in this Dallas situation?

Mr. FROST. Well, I do know that the State of Texas has been involved from the beginning, this particular case near the lead smelters we are talking about now, not the other site in my district, and that the Texas Air Control Board, as I mentioned, has been very concerned and is in the middle right now of attempting to seek enforcement proceedings against one of the smelters.

Now, in terms of the broader question of the soil, I am not certain. I cannot answer with specificity as to whether any of the other State agencies have looked at the soil problem.

Mr. MOLINARI. I have no further questions, Mr. Chairman. But I do think it would be important to the committee that we do ascertain the State evaluation of the site.

Sometimes there is great variance between the State's evaluation and the Environmental Protection Agency's evaluation. I think that is important information we should have before us.

Mr. Frost. It may well be. If I am able to find any information to that effect, I will be happy to provide it to the committee.

It may be the State of Texas has already done this.

Mr. Molinari. Thank you. We would appreciate it.

Mr. Levitas. Thank you.

Mr. Howard.

Mr. Howard. Thank you, Mr. Chairman.

Mr. Frost, I just wish to congratulate you and thank you for your appearance here; and also, to commend you for the long-time effort you have made concerning this problem. It not only shows the work of a fine Congressman, but a true representative of his people.

We do hope that with the information we will gather from you and your constituents who are here, that we may find some rapid relief as a result of these hearings.

Mr. Frost. Thank you, Mr. Chairman.

Mr. Howard. Thank you very much.

Mr. Levitas. Mr. Snyder.

Mr. Snyder. I just want to thank the gentleman for appearing and giving us this chronology of events. He has been very helpful in laying out the fact that EPA was well aware of the problem.

I appreciate it. Thank you for being here.

Mr. Levitas. Mr. Andrews.

Mr. Andrews. Thank you, Mr. Chairman. I just wanted to thank Congressman Frost for appearing before our subcommittee today.

This seems to be a recurring problem across this country. Also being a Congressman from Texas, in the Houston area, we, too, have serious toxic waste problems, and have been frustrated.

I think the documentation you have presented to us and the record of your actions are most commendable and will really assist this committee in making progress toward being sure that, first of all, toxic dump sites in our State of Texas are cleaned up, and second, that the laws of the Congress are carried out by the Environmental Protection Agency itself are carried out. Thank you.

Mr. Levitas. Thank you.

Mr. Clinger.

Mr. Clinger. No questions.

Mr. Levitas. Dr. Rowland?

Dr. Rowland. Thank you very much, Mr. Chairman.

Mr. Forst, thank you very much for being here. I believe according to this statement that you began to strongly focus attention on this problem in the fall of 1981. I commend you on doing that.

So many times there are things that just get by us. But they did not get by you.

Mr. Chairman, it is going to be very interesting to me to learn when children first began to manifest symptoms of lead poisoning in reference to the testimony that you have given here indicates.

Thank you for being here.

Mr. Levitas. Thank you, Dr. Rowland.

Congressman Frost, again, our appreciation to you. I know that you and your staff will be closely following the proceedings of today and subsequent events.

The important thing is to—and I am sure you know this, because you have expressed it to me—the concern for the people who live in that area and their health and that of their children.

Thank you.

Mr. FROST. Mr. Chairman, I, again, want to express my gratitude for the role of this subcommittee and ultimately to this full committee, because each of us in our own districts encounters problems like this from time to time.

The forum that you are providing and the attention you are focusing on this matter, and it richly and well deserves that attention, I think will ensure we will have some action in this particular case. I thank you for that.

Mr. LEVITAS. Thank you.

The next witness will be a panel from Dallas, TX, primarily focusing on just exactly what was occurring in this area near the smelting plants. The panel consists of Mrs. Patricia Spears from Dallas, TX; Ms. Lois Cross from Dallas, TX. They are accompanied by Ms. Jane Saginaw, an attorney with Frederick M. Baron & Associates of Dallas; and Mr. Steven Wodka, director, Health Effects Research, Frederick M. Baron & Associates, Dallas, TX.

We are extremely pleased to have you here today to share with us your experiences and the information that you have first-hand what was occurring in this area.

As is our practice before this subcommittee, being an investigative subcommittee, all testimony is taken under oath. So at this time I would like to ask all four of you, if you would, please raise your right hand.

[Witnesses sworn.]

Mr. LEVITAS. Thank you. It is my understanding that, Mr. Wodka, you are going to introduce the panel.

TESTIMONY OF PATRICIA SPEARS, DALLAS, TX; LOIS CROSS, DALLAS, TX; JANE SAGINAW, ESQUIRE; AND STEVEN WODKA, DIRECTOR, HEALTH EFFECTS RESEARCH, FREDERICK M. BARON AND ASSOCIATES

Mr. WODKA. Yes, Mr. Chairman. Our firm represents 200 children in West Dallas and South Oak Cliff in their claims against the three smelting companies there. We are here accompanying Ms. Spears and Ms. Cross who have worked very closely with our firm.

First, on my far right is Jane Saginaw, attorney in our Dallas office. Next to Jane is Ms. Lois Cross, who is social education director for the West Dallas Boys Club. To my immediate right is Ms. Patricia Spears, who is a resident and mother from the West Dallas community.

Ms. Spears will lead off.

Mr. LEVITAS. Thank you, Mr. Wodka. Before Ms. Spears testifies, I want to point out to my colleagues that it is my understanding as you have indicated that there is litigation now involving Ms. Spears and others in the neighborhood with respect to the smelters, and there may be some questions which are pertinent to that litigation, the answers to which would be considered confidential in nature, work product during the preparation of litigation.

In the event there are such questions and answers, we will make arrangements to receive that testimony in executive session. I just wanted to alert my colleagues of that.

Ms. Spears, first I want to thank you very much for coming from Dallas to help us do our job today. We are looking forward to your testimony, knowing that you have had first-hand experience, and would like for you to share with us your knowledge of what was going on, what the problems were, and help us understand as best we can just what was happening to you.

Ms. Spears.

Ms. **Spears**. Thank you, Mr. Chairman.

I would also like to thank you all for inviting me here today. I would like to start, first of all, by stating that I am a resident of West Dallas. I have been in West Dallas all my life.

I was raised on Westmoreland, about six blocks from this smeltering plant. From my childhood, I have experienced illnesses. My family members have. My sister has had numerous things on her legs.

Another sister had terrible skin rashes, had to have oxygen all the time due to respiratory problems. I am raising my children there now.

I am experiencing horrible situations with my children. My oldest son is now 14. He has been in and out of doctors' offices and hospitals since 3.

He was initially diagnosed for having some form of seizure, learning disabilities. I have never been told what form of seizures, what form of learning disability.

He has been in and out of the hospital. He has black-outs, chest pains, respiratory problems, leg aches and pains, along with three of my other children, severe headaches, stomach pains to the point where it will double them up in knots.

Headaches to the point where they can't stand for anyone to walk in the next room. I have had problems with the children in school as far as educational-wise.

They were in Dallas independent school district. I was called in, told that my son had serious problems, he could not learn.

He was placed in special education. I was not satisfied with that. I had him taken out of special education. He still could not keep up with his regular grade.

My other children, as they started school, they were having problems keeping up with their grades. I was paying tutors in the afternoons.

I have received help from the Boys Club with tutoring with my children. I was called in again when my 14-year old was in fifth grade, told that he needed to go back in special education, he just could not learn.

At that time, my second son, my baby son, was also experiencing problems in school. His attitude changed. His behavior problems, learning problems, they were having severe problems even keeping him still in class.

At that time I placed the children in Catholic school. They were there for 2 years. I worked very closely with the sisters there.

They did their very best, but I was called in again and told that they needed to be in some place like the Scottish Right, or some place, because they needed some special attention, that there was just no way that their attention span could, you know, there is no way, you know, they could help them.

I am still experiencing problems. I am at a point now where I am trying to get them in through Scottish Right, or some school. My 8-year-old, my baby, has a limp now. She limps with her right leg and it is getting increasingly worse.

I had a conference with her teacher yesterday. She said that she was doing better, but she is falling back in a lot of her subjects.

The same thing with the other children.

Thank you.

Mr. LEVITAS. Thank you, Ms. Spears. We want to ask you some questions.

But before we do that, perhaps, Ms. Cross, if you could describe the experience that you had in your work in this area to relate to us what conditions were, where the children were playing, and just how it related to this whole question of smelting facilities.

Ms. CROSS. Thank you, Mr. Chairman.

I have been working at West Dallas Boys Club for 3 years now. I have been working with the education program at the boys club, and where we have an afternoon tutoring program for the kids who have—need help in their grades.

I have noticed a decrease in the grade level, where, as concerned parents that come to me and ask me about their kids, and they wanted help for their child because their grade level just dropping too fast, you know.

Their learning ability was not up to par. They wanted help. They need help. So what I did, I took it on my own to go to the neighborhood schools and talk to the principals and the teachers and the counselors and set up some type of program on my own to try to come out and to enhance those kids' learning ability.

What I did, I came back to the boys club and talked to my immediate supervisor and we set up an afternoon tutoring program for the kids.

Those kids are in there from 2:30 to 6:30 at night. That way they are getting help in the subjects that they need help in.

A lot of them is having trouble in science and math and different subjects like that. But our disability to the kids is that they come in at 2:30 and they stay there for 6:30, and the mothers pick them up at 6:30. They do not get a chance, opportunity, to go back in the recreation part to play in the activity.

Why? Because they are there in the afternoon, all day in school. They have books there at school. Then in the afternoon they come back to the boys club.

They are just in tutoring all day. What we thought would be best for them to do, and we had this tutoring program to try to make up their level, grades level, but I have noticed that they are still needing tremendous help in the learning ability.

Also, parents was concerned about the absentees, that their kids was missing from the school. They were concerned because each week, maybe 3 or 4 days out of the week they were sick.

They cannot go to school. They would go to school. They would have to leave their jobs, go pick them up and take them to the doctor. They was complaining about different things like that.

The principal, they was saying that they cannot miss these X number of days out of school. And the kids, I know also the kids in the tutoring program in the afternoon they were always complaining about fatigue.

They was tired. Their stomachs ached, dizziness. They just had different problems. Also, that we had to close down at the boys club.

They were condemned on the playground. We could not go outside with the kids for a couple months because of the high level lead at the boys club. We also closed our pool, swimming pool down, that we just opened back up the first of the year, because the high level lead was found in the swimming pool.

At that time RSR, they did remove the soil in the playground and they did the filter work at the boys club in the swimming pool. Now we are able to go back into the swimming pool and run activity in the pool.

But that time the kids were condemned to no outdoors activity at all.

Mr. **Levitas**. Thank you, Ms. Cross.

Ms. **Cross**. Thank you.

Mr. **Levitas**. Ms. Spears, were the type of conditions that you have described about your children, their health and their problems, that you knew of, in the families of others in your neighborhood?

Ms. **Spears**. Oh, most definitely. I could name, oh, tremendous amounts of families that is having the same problem, or similar problems, or something even worse than the problems that I am having, you know.

Mr. **Levitas**. When did you first begin to get some thought that this problem that your children and others in your neighborhood were experiencing related to the lead poisoning?

Ms. **Spears**. After my baby daughter, Demetria, was tested, had the first initial test. It wasn't until then when I started suspecting.

Mr. **Levitas**. Approximately when was that?

Ms. **Spears**. It was in 1981. The first initial blood test that the city started.

Mr. **Levitas**. Have all of your children had blood tests?

Ms. **Spears**. Yes, they have.

Mr. **Levitas**. Did they find levels of lead in their blood?

Ms. **Saginaw**. These matters are going to come out in discovery in the lawsuit as they are now pending. It is not public knowledge right now. We will be glad to provide you with Mrs. Spears, her children's blood levels, and all of that information when it comes out in discovery.

Mr. **Levitas**. Do you know, Ms. Spears, have you been told whether the types of symptoms your children experienced are similar to the types of symptoms that persons suffering from lead poisoning experience?

Ms. Spears. Yes, I have.

Mr. Levitas. Now, Ms. Cross, you mentioned the fact that there were high levels of lead in the soil on the playground areas of the boys club. When did you find that out? What information did you receive about that?

Ms. Cross. Well, I didn't find it out. My boss found it out. I am just Education Director there, and I don't know exactly when they found that out. All I know, they did replace the soil there.

Mr. Levitas. About when did that occur?

Ms. Cross. I believe in 1982; last year, sometime.

Mr. Levitas. Now, I am interested in finding out about the conditions in the neighborhood, when it became known in the neighborhood that there were high levels of lead in the soil, and that children were manifesting these symptoms which were similar to that suffered by people with lead poisoning.

Was there any community concern as a community?

Ms. Cross, do you want to tell me about that? What was going on?

Did anybody complain to the city, or to—Ms. Spears, do you know?

Ms. Spears. Yes. There was great concern throughout the community, all of the communities that were involved. There was the West Dallas Involvement Committee formed. We went to work on trying to find out what we could do to help, what could we do to get the problem solved.

We spent numerous hours at city hall speaking with different officials there. We assisted and when the blood lab test screening program started, we volunteered.

We assisted in getting people to the testing sites. I worked on the spot volunteering in numerous of the lead screening programs along with other members of the communities. We did everything we knew to do, trying to find out what could we do as home owners, what could we do as parents and as a community to get the problem solved.

Mr. Levitas. Did you ever have any dealings with people from the U.S. Environmental Protection Agency?

Ms. Spears. Some, yes.

Mr. Levitas. And the city officials of Dallas, who were the people you talked to at the Dallas City Hall?

Ms. Spears. At that time, Assistant City Manager Levi Davis was in charge.

Mr. Levitas. Was there any concern in the community that there weren't enough steps being taken to cleanup the problem?

Ms. Spears. Yes, and it still is. A great concern.

Mr. Levitas. Would you explain a little bit about how people, you and others that you spoke to, felt about the fact that there was nothing being done, or not enough being done?

Ms. Spears. We were very unhappy about it. We thought after the first initial blood testing and the soil and samples were taken, that after the study came back, you know, that something would definitely be done about the problems.

We found that then they had to go back and do—they said those studies were incomplete. They had to go back and do scientific tests.

Then we finally were doing nothing but just waiting, waiting, waiting. There is nothing been done about it. I have, in talking to members of the community, there are families and senior citizens who are still afraid to come outside or to work their yards, work their gardens.

We love our community. We want to keep our community, and we want the problem straightened out. There is general concern throughout all the communities that were involved.

I have worked very closely, not only in West Dallas, but in the other communities where they had the similar problem. I have talked to numerous people about it, and we were deeply concerned about our children's health, about our property, and about just what is going to happen to us and to our community as a whole.

Mr. LEVITAS. You talked about the illnesses of your children, and you said that your daughter, I believe it was, had her blood tested in 1981. Then you found out that there were going to be another set of tests run.

Did you or did others involved in this express concern that after the first round of blood tests, something ought to be done?

Ms. SPEARS. Yes, I did, on numerous occasions. I really did. I kept going, I have asked. There was one other council person that was deeply concerned, deeply sincere. She constantly asked, she constantly talked about it, stayed on top of it, working directly with the assistant city manager.

She did everything that she knew to do. She called meetings, town hall meetings at the Martin Luther King Center. We would go there. We would have to go there to try to ascertain information.

That is where EPA came out and talked to all of us and was, you know, tried to let us know what was going on.

Mr. LEVITAS. You said earlier that there were people in the community with their concerns who were afraid to go out and work their gardens and work in the yards; that parents were concerned about their children.

Would you say that there was a fear in the neighborhood?

Ms. SPEARS. A definite fear. I am afraid myself. I have had to see my children suffer so.

I have been in and out of doctors'—and like I said, hospitals, with my children since they were infants. They were hospitalized on numerous occasions for different things.

They have been treated for anemia. As a matter of fact, I have been treated for anemia. I have just seen a lot. I was raised there, and I have seen a lot of things, questions that could not be answered.

I didn't have the answer for it. It wasn't until, like I say, in 1981 when we were thinking that it might be—the illnesses and things might be from the problems that, you know, has come out about the lead situation.

Mr. LEVITAS. Thank you, Ms. Spears. I may have additional questions.

At this time I would like to recognize the gentleman from New York.

Mr. MOLINARI. Thank you, Mr. Chairman.

Mrs. Spears, I want to thank you for coming before us to help us wrestle with this problem.

Ms. **Spears**. Right.

Mr. **Molinari**. I just have a few questions. In relationship to where you live, which of the three, RSR, Dixie, or National Lead, are you closest to?

Ms. **Spears**. RSR.

Mr. **Molinari**. How far is your home from RSR?

Ms. **Spears**. About 8, maybe 10 blocks now.

Mr. **Molinari**. Would you happen to know whether you are downwind of that plant in the normal wind pattern?

Ms. **Spears**. Yes, sir.

Mr. **Molinari**. How about Dixie Metals and National Lead, how far are they from your home?

Ms. **Spears**. Pretty good piece.

Ms. **Saginaw**. Well, it is about—I would say maybe 5 or 10 miles away.

Mr. **Molinari**. Five miles?

Ms. **Saginaw**. Five or ten miles.

Mr. **Molinari**. Five or six miles?

Ms. **Saginaw**. I really don't know.

Mr. **Molinari**. OK. Ms. Spears, how many children do you have?

Ms. **Spears**. I have four.

Mr. **Molinari**. The ages of your children, please?

Ms. **Spears**. Fourteen, thirteen, eleven, and eight.

Mr. **Molinari**. When was it that you first started experiencing health problems with your children?

Ms. **Saginaw**. These questions could get into a statute of limitations problem or something with the litigation. Exact dates and that sort of thing, Ms. Spears doesn't want to go into in great detail.

I think she said she has had problems with her kids all her life.

Mr. **Levitas**. Pull the microphone closer to you.

Ms. **Saginaw**. Questions of specific dates could have an effect on the pending litigation. Ms. Spears does not want to talk precisely about specific dates.

Mr. **Molinari**. These are continuing symptoms, are they not?

Therefore, wouldn't the statute of limitations continue to run since the symptoms are continuing?

Ms. **Saginaw**. That is a continuing question that will have to be decided in the litigation. That is why Ms. Spears isn't——

Mr. **Molinari**. We would like to get some frame of reference here so we know whether this is a comparatively recent problem, or one that has existed for many years so that we can adequately deal with the subject matter of the hearings. Now, maybe Ms. Spears is not the one to answer that question, if counsel doesn't want her to.

But for us to be able to proceed intelligently, I think we have to get some fix on the time.

Ms. **Spears**. In 1981 was when the problems were worse. That is when I had serious, severe problems.

Mr. **Molinari**. You experienced problems you say. You mean yourself or your children?

Ms. SPEARS. With my children.

Mr. MOLINARI. You mentioned anemia. Have they been tested for anemia?

Ms. SPEARS. I stated they have been treated for anemia.

Mr. MOLINARI. Is that all your children?

Ms. SPEARS. Yes.

Mr. MOLINARI. That is since 1981, also?

Ms. SPEARS. That was before 1981.

Mr. MOLINARI. Excuse me. Before 1981?

Ms. SPEARS. Yes.

Mr. MOLINARI. Did the doctors tell you there were health problems in the urinal tract or the gastrointestinal tract? I think you have mentioned before in your testimony they had stomach problems.

Ms. SPEARS. Yes.

Ms. SAGINAW. Mr. Chairman, if I may, I believe Ms. Spears, when she came to testify, she wanted to come and talk about the general problems that she was having. We were told that she wouldn't be asked real specific questions about when and what. This is all matters of the litigation that will come out. We will provide you, we will be glad to provide you with doctors' diagnoses and neurological——

Mr. MOLINARI. I am not aware of those guidelines. So if there was such a guideline, I would ask the chairman for direction.

Mr. LEVITAS. Can you provide for us the record copies of the physicians' diagnoses of Ms. Spears' children?

Ms. SAGINAW. Absolutely.

Mr. LEVITAS. Without objection we will receive them.

[The information received follows:]

The University of Alabama in Birmingham
School of Social and Behavioral Sciences
Department of Psychology
205/934-3850

NAME: DEMMINGS, Demitria

SEEN: 12/14/82

AGE: 8

DOB: 10/5/74

EDUCATION: 2

Demitria Demmings was seen for neuropsychological evaluation on December 14, 1982. At that time a battery of psychological-behavioral measures was administered including the Wechsler Intelligence Scale for Children-R, the Wide Range Achievement Test, an Aphasia, Sensory Perceptual and Motor Examination, and the Reitan Indiana Neuropsychological Test Battery for Children and associated memory and language procedures.

The Wechsler Adult Intelligence Scale for Children-R was administered and Demitria obtained Verbal and Performance IQ Scores that fell within the dull normal range (VIQ = 88, PIQ = 84). Her general pattern of performance was mildly disrupted with respect her verbal acquisitional competencies and markedly disrupted with respect to visuo-spatial and problem solving skills suggesting a degree of cognitive inconsistency that goes beyond the normal limits and may well serve as a disruptive focus with respect to utilization of cognitive skills in a variety of contexts.

The Wide Range Achievement Test was administered and Demitria obtained Grade Placement Scale and Percentile Scores as follows: Reading Recognition 2.8, 93, 32; Spelling 3.1, 97, 42; and Arithmetic 2.5, 85, 16. These suggest for a young lady currently in the second grade that she is making at the current time a degree of academic progress essentially commensurate with expectations for her grade level and her psychometric development. As this test tends to represent something of an overestimate of actual academic placement in the very early years, however, monitoring of this continuation of adequacy would be important particularly in the context of some degree of disruptiveness of cognitive process noted from the psychometric measurements referred to above.

A Sensory Perceptual and Motor Examination was administered and no significant deficits in this area were identified. Aphasia Examination revealed mild immaturities of both a linguistic and visuo-spatial nature.

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The Reitan Indiana Neuropsychological Test Battery was administered and Demitria demonstrated, in highly structured and supportive situations, good capacity in the areas of problem solving and general mental efficiency. She did, however, experience significant difficulties in certain new and unfamilar situations and demonstrated tendencies toward mild to moderate memory difficulties of a nonlanguage as well as a language type in tasks with considerable degree of either unfamiliarity or high mental demand.

From a neuropsychological point of view, Demitria's general pattern of performance is mildly inconsistent possibly suggesting some degree of generalized developmental disruption which is likely to represent a disadvantage for her in a variety of situations. Her general cognitive development appears to be low average to average suggesting the basic potential for normal functioning outside those areas of disruptiveness. For that reason, it would be worthwhile to monitor her academic and other developmental progress to see to it that no additional deficits occur and that remedial intervention occur in those areas where she is experiencing difficulties if such should be the case. If it is not the case then monitoring would appear to represent the appropriate intervention strategy in this case. Certainly this degree of cognitive disruptiveness should also be monitored to determine whether or not it represents developmental immaturity which is likely to spontaneously improve or whether it represents a degree of deficit and impairment requiring additional intervention activities in the future.

Thomas J. Boll

Thomas J. Boll, Ph.D.
Professor of Psychology
Pediatrics and Neurological Surgery

Director, Medical Psychology Program

TJB/rg

UAB
The University of Alabama in Birmingham
School of Social and Behavioral Sciences
Department of Psychology
205/934-3850

NAME: DEMINGS, Patrick

SEEN: 8/5/82

AGE: 13

DOB: 10/3/69

EDUCATION: 6

Patrick Demings was seen for neuropsychological evaluation at which time a battery of psychological-behavioral measures was administered including the Weschler Intelligence Scale for Children-R, Wide Range Achievement Test, an Aphasia, Sensory Perceptual and Motor Examination, Halstead Neuropsychological Test Battery for Children and associated procedures, Rorschach Technique and a clinical interview.

The Weschler Intelligence Scale for Children-R was administered and Patrick obtained Verbal and Performance IQ scores that fell within the average to borderline range (VIQ 94, PIQ 78). Patrick demonstrated moderate to significant inconsistency in general cognitive function and problem solving capacity and while his general verbal acquisition and ability to utilize past learning and experience were not unduely disrupted his visuo-spatial and problem solving and motor manipulatory problem solving skills were significantly uneven and suggestive of a young man who on a day to day basis is likely to be quite cognitively inconsistent utilizing data from this aspect of this examination alone.

The Wide Range Achievement Test was administered and Patrick obtained grade placement, standard and percentile scores as follows: Reading Recognition 6.8, 94, 34; Spelling 2.2, 66, 1; and Arithmetic 3.6, 70, 2. It is quite clear that despite the fact that Patrick is close to being competitive and psychometrically appropriate with respect to word recognition skills his skills in the areas of spelling and arithmetic computation are sorely deficient and likely to represent a significant disadvantage for him in making other than the most rudimentry types of progress in a normally progressing 6th grade setting.

Additional language examination reveals marked deficits with spelling, receptive language comprehension and verbal memory. Here too, the suggestion is that Patrick will require additional educational aid, individual tutoring or resource help in order to keep him from falling significantly behind his normally progressing classmates. Sensory Perceptual Examination reveals bilateral tactile perceptual immaturities of a mild nature, reasonably adequate motor speed, strength and psychomotor coordination bilaterally.

DEMINGS, Patrick
Page 2
8/5/82

The Halstead Neuropsychological Test Battery for Children was administered and Patrick demonstrated at least mild learning inefficiency on a complex test of problem solving in a new and unfamiliar environment. Despite the structure and support of this situation he demonstrated a degree of inconsistency not surprising in light of his performance on psychometric measures in suggesting again a degree of inefficiency that is likely to be disruptive to day to day performances including those of an academic nature.

From a neurodevelopmental point of view, Patrick appears to be a young man with neurobehavioral and neurodevelopmental impairments and disruptions that are significantly negatively influencing his overall cognitive ability ability to utilize his mental capacity adequately in general terms and with specific reference to academic functioning. This is true despite the fact that in Verbal IQ terms he is at least average, but nevertheless, experiences difficulty in other areas of mental functioning.

From a psychological point of view, Patrick appears to be a young man with a relatively poor self concept who is moderately anxious, angry and experiences difficulty in controlling his emotions. He appears to be somewhat overwhelmed by his feelings of psychological uncertainty and anxiety about physical and psychological well being. While Patrick has a healthy need for affection and emotional closeness he appears to be markedly anxious about the thought of high levels of emotional closeness and strong emotional expression it would be entirely appropriate not only for Patrick to experience special educational aid to reinforce those mental abilities which he does have and to aid him to make far more academic progress than has been possible to date but also to aid him with his marked psychological anxiety and fears about his physical and social well being which cause him to border on the moderately distressed and subjectively depressed. Certainly this represents yet an additional deficit to his day to day capacity which when coupled with his very real cognitive inconsistencies and inefficiences further compromise his ability to use his mental capacities in a normal and adequate fashion.

Thomas J Boll

Thomas J. Boll, Ph.D.
Professor of Psychology
Pediatrics and Neurological Surgery

Director, Medical Psychology Program

TJB/rg

Mr. MOLINARI. Thank you.

Ms. Spears, let us leave the dates out, because of the pending litigation. But when you started having these problems, did you go to a local doctor first and have him check your children out?

Ms. SPEARS. Yes, several. Yes, clinics, doctors, hospitals.

Mr. MOLINARI. Is the clinic attached to a hospital?

Ms. SPEARS. Well, they have clinics in West Dallas where we live. Then there is the hospitals for emergency, at night, and more serious problems.

Mr. MOLINARI. Was this frequent? I mean did you go there often?

Ms. SPEARS. Yes.

Mr. MOLINARI. Did they refer you to the city health authorities?

Ms. SPEARS. No, never.

Mr. MOLINARI. Did you ever contact the city of Dallas, the health department, complaining of this problem?

Ms. SPEARS. No. I didn't know to. I didn't know what was wrong with the children. They was just sick, so I was carrying them to the doctor.

Mr. MOLINARI. Nobody suggested to you that perhaps the health department of the city of Dallas should look into the matter?

Ms. SPEARS. No. As I stated before, it wasn't until 1981 when I realized that these could be——

Mr. MOLINARI. I understand. The reason I ask this is that usually what happens when you have a situation like this, in the first instance, people will generally be referred to the local health agency. I would assume the city of Dallas has a rather sophisticated health department. Where there is an epidemiological problem like this that affects so many children in the area, normally they would be the agency that would first become involved.

How about the State? Was the State involved in any way?

Ms. SPEARS. Not to my knowledge. Let me explain something. I was stating about my children's health problems that I have experienced with them from infant, up, you know. I was just carrying them to the doctor. They were sick. And my natural, as a mother, I would just carry them to the doctor. And, you know, I had no way of knowing that I, you know, needed to look in, because I wasn't aware of the problem.

Mr. MOLINARI. Ms. Cross, do you know whether, with the problems that you are experiencing at the school, anybody from the Dallas City Health Department came to do a check? Were they called in, or did they come to the school to run any checks?

Ms. CROSS. Only thing I am aware of is that the EPA, we did hold a testing site at the Boys Club to do blood level tests.

Mr. MOLINARI. EPA.

Ms. CROSS. EPA.

Mr. MOLINARI. But you don't know of any involvement on the part of the city of Dallas.

Ms. CROSS. It was the city that we held the testing site for.

Mr. MOLINARI. I can't hear very well up here.

Ms. CROSS. It was the city of Dallas that we was holding the testing site at the Boys Club to do the testing, blood level testing.

Mr. MOLINARI. The city of Dallas did?

Ms. CROSS. Yes.

Mr. **Molinari**. Mr. Chairman, I think we should get the data that may exist in the city health department for introduction into the record. Thank you. I have no further questions.

Mr. **Levitas**. Thank you. I think your suggestion is a good one. Also whatever is available from the State agencies as well.

Dr. Rowland?

Mr. **Rowland**. Thank you very much, Mr. Chairman, and Mrs. Spears and Ms. Saginaw, Ms. Cross, Mr. Wodka. I thank you all very much for being here. I know coming to Washington is not an easy thing to do. I am grateful that you are here to help shed some light on this problem.

I want to go back a little further. You mentioned that you grew up in West Dallas——

Ms. **Spears**. Yes.

Mr. **Rowland** [continuing]. When you first started talking a few minutes ago.

Ms. **Spears**. Yes.

Mr. **Rowland**. How close did you live to the smelters when you were a child growing up there?

Ms. **Spears**. Very close. I guess it is about four blocks or so, six blocks. No more than six blocks.

Mr. **Rowland**. How many brothers and sisters do you have?

Ms. **Spears**. I have five sisters.

Mr. **Rowland**. You have five sisters.

Ms. **Spears**. Right. I have five sisters. But there were five of us living in that house. My other sister is grown.

Mr. **Rowland**. When you were growing up, there were——

Ms. **Spears**. Five of us. Five children.

Mr. **Rowland**. Five children living in the house. You mentioned some of the problems, I believe, that your sisters had when they were growing up. I think you mentioned some skin rashes?

Ms. **Spears**. Yes.

Mr. **Rowland**. And some problems with legs, having some operations on the legs?

Ms. **Spears**. Yes.

Mr. **Rowland**. Were you carried to the doctor then? Did your parents carry you to a doctor when you were growing up about the problems that you were having?

Ms. **Spears**. Yes, and we have all been hospitalized for several problem things, problems we were having then.

Mr. **Rowland**. Were blood levels for lead ever done on you when you were a child, do you recall?

Ms. **Spears**. No, never.

Mr. **Rowland**. Did any physician or any doctor as you recall mention to your parents that the problems you were having may have been related to where you were living?

Ms. **Spears**. Never. No. Not to my knowledge.

Mr. **Rowland**. Never anything said about that at all?

Ms. **Spears**. No.

Mr. **Rowland**. Can you describe the skin rash? Do you recall, and if you don't, that is fine.

Ms. **Spears**. I recall. With my sister it would break out, and it was like something would, you know, eat away at the flesh in her arms, her legs, in her face, her hands. Pretty much all over. Her eyes would swell.

Mr. Rowland. Was it just one sister that had this problem?

Ms. Spears. Well, they said it was allergies. There was one sister that had the rash. But the allergies, we all had that problem.

Mr. Rowland. Did all of you have skin rashes?

Ms. Spears. Not to that extent.

Mr. Rowland. I see. Do you ever recall whether there was anything unusual about your gums when you were children growing up?

Ms. Spears. They would bleed all the time.

Mr. Rowland. Do you recall anything about the color of your gums? Were they the normal color of other people's gums, do you recall?

Ms. Spears. No, they were darker.

Mr. Rowland. Darker?

Ms. Spears. Yes.

Mr. Rowland. Could you attach a color to that darkness? If you can't it is all right.

Ms. Spears. Bluish-black-like.

Mr. Rowland. I see. Other children didn't live in this area, their gums were not that color, do you recall? Or you don't recall?

Ms. Spears. I don't know.

Mr. Rowland. You did realize, though, that your gums were somewhat different?

Ms. Spears. Yes. They bled all the time and they would swell.

Mr. Rowland. What about your children? Did you ever notice anything about their gums that may have been different from other children, or about the same?

Ms. Spears. Well, I carry them to the dentist regular, and he didn't mention it. No, I hadn't noticed it.

Mr. Rowland. I believe, Mr. Wodka, you said there are 200 children that you are representing now?

Mr. Wodka. Yes, sir.

Mr. Rowland. Are there 200 children that have had elevated lead blood levels?

Mr. Wodka. Let me refer that to Ms. Saginaw.

Ms. Saginaw. There are 200 children that have come to us with different kinds of complaints that they feel might be related to lead, and we are looking into those. We are looking into all the different problems in the neighborhood.

Mr. Rowland. Have these children had blood levels done?

Ms. Saginaw. Most of them have.

Mr. Rowland. Could you tell me whether or not they have had elevated blood levels?

Ms. Saginaw. Some of them did, and some of them didn't. Some of them were tested by the city and the EPA, and some of them weren't. Everyone is in a different position out there right now. But we are investigating into all the different claims.

Mr. Rowland. Mr. Chairman, thank you very much. Thank you very much for your testimony.

Mr. Levitas. Thank you.

Mr. Snyder.

Mr. Snyder. Ms. Saginaw, you are counsel for these people in the lawsuit?

Ms. Saginaw. That is right.

Mr. Snyder. And Mr. Wodka, is that a correct pronunciation?

Mr. Wodka. Yes, sir.

Mr. Snyder. Are you with the law firm?

Mr. Wodka. Yes, I work with the law firm.

Mr. Snyder. Are you a lawyer?

Mr. Wodka. No; I am director of health effects.

Mr. Snyder. What is your professional background?

Mr. Wodka. I am a law student right now. I don't have any professional medical——

Mr. Snyder. Could you give me your title again?

Mr. Wodka. Well, Mr. Congressman, when you are a law student working for a law firm and doing research you have to concoct a title.

Mr. Snyder. Well, it is kind of like being a Kentucky colonel, it doesn't mean much.

Mr. Wodka. That is for sure.

Mr. Snyder. I see by your card you are director of health effects research.

Mr. Wodka. I have worked in this field for 13 years, mainly in occupational health areas.

Mr. Snyder. For the record, based upon your research, what are the symptoms of lead poisoning or excessive lead in your system?

Mr. Wodka. Well, generally, Mr. Snyder, I think a number of the things you have heard today from Ms. Spears and Ms. Cross, the problems with the gums that Congressman Rowland brought out, the stomach pains, the learning disabilities; the more serious permanent effects, which would be brain damage, which could be evidenced by the learning difficulties that you have heard here today. We could provide you, we could provide this committee with a much more comprehensive list.

I think it is shocking, the fact that—I think as Mr. Molinari was trying to get at, that this situation exists down there. But the State health department and neither the city, neither has done the comprehensive kinds of tests on the people in that community as they should have done.

Mr. Snyder. I think, as an observation, it was evidenced from Congressman Frost's testimony that the State air people were not moving until perhaps this committee got involved, gave them a little publicity and put the heat on.

Mr. Rowland. Will the gentleman yield?

Mr. Snyder. Sure.

Mr. Rowland. With reference to the symptoms, what I was pursuing a moment ago, in chronic lead poisoning, a lead line develops in the gums. It is enlightening to me that she described the lead line in her gums. I am surprised that no physician suspected that when they were being treated as children. The symptoms of lead poisoning, chronic lead poisoning are variable. Neuropathy develop with it. Pains, very vague gastrointestinal problems, problems related to the urinary tract. The symptoms can be just almost innumerable in number. But there are certain things that indicate the possibility of poisoning. This lead line is one of those things.

Mr. **Snyder**. I guess it is highly unusual. But could I ask the doctor a couple of questions? How is lead normally absorbed into the body? Is it through the air, or through the mouth, or through the pores?

Mr. **Rowland**. Right, all three ways that you mentioned. As a matter of fact, one of the things that used to cause lead poisoning many years ago was the burning of battery boxes, and the smoke in the air. The children would inhale the smoke and get it that way. Lead in paint. We don't allow lead in the paint, lead on the cribs of children. Contact through the skin. So you can get it in many different ways.

Mr. **Snyder**. I guess the leaded gasoline you get by breathing the exhaust fumes?

Mr. **Rowland**. I don't know that that is a significant way to get it, not in my experience anyway. I am sure that this could be a problem in the urban areas where there is much exhaust in the air.

Mr. **Snyder**. I don't have any further questions, Mr. Chairman. I thank my colleague for his enlightening statements.

Mr. **Levitas**. Thank you very much. We appreciate the light you have been able to shed on this in giving us a picture of just precisely what was happening in that community, and the fears and the concerns which you have as people in the community and as parents. I hope that as a result of the efforts of this subcommittee, we will be able to help the appropriate governmental agencies address this problem in a much more prompt and effective manner than apparently has occurred up to the point where it is now.

Thank you very much, and we appreciate your being with us.

The next witnesses are a group of officials from the region VI office of the U.S. Environmental Protection Agency. They include Ms. Frances Phillips, deputy regional administrator, Mr. Allyn M. Davis, director of air and waste management, and Dr. Norman Dyer, who is chief, pesticide and toxics branch in region VI, U.S. Environmental Protection Agency.

If you would please come forward, and I will administer the oath to you.

And Mr. Richard Whittington, regional administrator of region VI, EPA.

[Witnesses sworn.]

Mr. **Levitas**. Thank you very much. We appreciate your being with us today to help shed some light on just exactly what happened in the matter of the Dallas lead smelting site. As you know, I am sure you have been briefed as to the testimony we received last week. Since this matter was being handled in region VI originally and primarily, we need to find out from your perspectives just exactly what went on, when you got started, and where it went to and why. So with that sort of general directions, perhaps, Ms. Phillips, would you be the appropriate person to begin to give us this history?

TESTIMONY OF FRANCES PHILLIPS, DEPUTY REGIONAL ADMINISTRATOR, U.S. ENVIRONMENTAL PROTECTION AGENCY, REGION VI, DALLAS, TX; ALLYN M. DAVIS, DIRECTOR, AIR AND WASTE MANAGEMENT DIVISION, U.S. ENVIRONMENTAL PROTECTION AGENCY, REGION VI, DALLAS, TX; NORMAN DYER, PH.D., CHIEF, PESTICIDES AND TOXICS BRANCH, U.S. ENVIRONMENTAL PROTECTION AGENCY, REGION VI, DALLAS, TX; RICHARD WHITTINGTON, REGIONAL ADMINISTRATOR, U.S. ENVIRONMENTAL PROTECTION AGENCY, REGION VI, DALLAS, TX.

Ms. PHILLIPS. Yes, Chairman Levitas. I would be glad to.

During the period of March 1981 until September 1981, I served as acting regional administrator of the Agency. In 1980, EPA, region VI, began what we called at that time an integrated toxic study of lead smelters in our region. That was partially a soil study.

I have with me Dr. Allyn Davis, who was responsible for the program at that time. I think perhaps from a historical perspective standpoint it would be helpful if he started off at this point.

Mr. DAVIS. My name is Dr. Allyn Davis. The regional office had a small toxics staff. As you are aware, the toxics and pesticides program are generally centralized product programs in Washington, but we do have a small staff in the regional office. We were looking for a group in the regional office to work together to identify potential problems in the region that existing statutes or Federal, local, State programs weren't addressing. This task force was comprised of members from each of the divisions, and developed a strategy for the regional office for toxic chemicals ranking the toxic chemicals of interest.

The final report identified six chemicals of interest, particularly in the region. Lead came out No. 1. The reason we were interested in lead and the ranking criteria were essentially as I pointed out, the gaps in the regulations, the potential problem, population exposure and information available both in the literature and in the environment.

At that time we began letting a series of contracts in the region to take a look at environmental factors. We had studies in Corpus Christi, Houston, Shreveport, Baton Rouge, Frisco, TX, and Bartlesville. At the beginning we were primarily looking at elevated levels of lead in the soil, tap water, and dust in the home. Those studies were started in the Dallas area in the fall of 1980.

Our first priority on letting the contracts would be to a State agency such as department of health if it was interested. If not, the local university, and only as a last resort a consulting firm, because we wanted local participation early on.

In the Dallas situation the contracts went to the University of Texas at Arlington. The work was done from October through December 1980. And the report was provided to the regional office, I believe, in February. As opposed to several of the other studies that were underway, the Dallas studies showed elevated levels of lead in the environmental around two locations. One was a western Dallas smelter location, and the second was a south Dallas location where there were two smelters across the street from each other. One had shut down in 1979, the other was an active facility. When the report was provided to the regional office we tried to interpret the data.

In general, around one of the smelters, 25 percent of the samples were over 1,000 parts per million. At the other location, 30 percent of the samples were over 1,000 parts per million.

Mr. **Levitas**. What is the significance of 1,000 parts per million?

Mr. **Davis**. We also did a reference area, to give you a perspective. I believe the reference area had 54 parts per million. The average value around one of the smelters was about 500, the average was—around the other was about 700. But what was really of concern were the numbers above 1,000 parts per million. The reason we were concerned was obviously they were higher than background, but also the Agency had an internal alert level. That if 10 percent of the samples were over 1,000 parts per million that was an abnormality and indicated the need for further research, further study to find out if there is an environmental or health problem.

At that time we started to consult with our headquarters group, reviewing the work that was done in Dallas and asking for interpretation, working with the city of Dallas, the State health department trying to find health data that may give us an indication of a problem in the community. The city had extensive blood screening data from the 1970's. Unfortunately, very few of the 7,000 or 8,000 children tested were within the 2-mile circle that we were concerned with. As a matter of fact, I think only 27 children were within that 2-mile circle.

And there was also at that time some question of the validity of the city's data because of the method they utilized in taking the blood samples. So we had elevated levels in the environment and a very confused situation regarding the medical data that was available to us. During this time period we were meeting with the city, myself or my staff, maybe one day or twice a week to try and find out, did we have a problem and what the appropriate action should be.

Within the regional office we were also consulting on was there an environmental problem and what action should be taken. After consultation with a variety of individuals in the regional office from a variety of divisions and also our headquarters group, we generally came to the conclusion that levels above 1,000 parts per million should be addressed, and there might be a problem below 1,000 parts per million. We were not sure. And there was an indication for additional testing to confirm if there was a medical problem caused below 1,000 parts per million in the soil.

In late May I had a commitment from the city of Dallas that, if the data was inconclusive, they would go forward with a medical screening program for the area to try to answer the question: Is there a problem?

Let me backstep to late April. At that time we called the companies in individually, I believe——

Ms. **Phillips**. Let me stop you right there, if you don't mind, on that one, because I think I am probably the best person to speak to this because it wasn't a call from EPA to the companies to bring them in. I was initially contacted by the RSR Corp., approximately April 20 or April 21. Excuse me. April—before April 20, because on

April 21 at the company's request, EPA did have a meeting with the RSR Corp.

The RSR Corp. came in and said that they knew that we had received the results of the study. They knew that we were talking with State and local officials about what the study meant. They said they would like to have a copy of the study. Whatever the study said, they wanted to assure me and they wanted to assure EPA that they would do whatever it took to alleviate the problem. That was an oral commitment at that April 21 meeting initially.

In addition to that they presented EPA with a letter that put that initial general commitment in writing. It was at this first meeting with RSR Corp. that I believe we talked about our concerns, about the 1,000 parts per million level, and that was the level we were looking to as an alert level, that we felt like we would need to do additional sampling to determine to what extent we had a 1,000 parts per million problem and to what extent we might have additional problems in the area.

From the April 21 meeting with RSR Corp. we then called in the Dixie Metals Corp. and went over essentially the same information with them, and told them the initial results of the study. I didn't mean to interrupt, but I did need to correct that, Allyn.

Mr. Davis. Thank you. We did discuss the 1,000 parts per million alert level at the April meeting, but it wasn't until the meetings in late May that we explicitly indicated to the companies that we were requesting cleanup at 1,000 parts per million as well as additional studies. We also were preparing to release the report. We had several freedom of information requests from both the companies and the media, and were planning to release the report and our interpretation of the data and our next step approximately on June 1.

At the meetings with the companies on May 28, we also gave them our draft press release, and asked them to add any comment that they wished to make as to what they were going to do as a result of our studies, and asked for their comments back. We met in the regional office on Saturday, May 30, to go over the responses from the companies. At that time, RSR indicated that they would clean up at the 1,000 parts per million level, and would participate in any additional studies. Dixie Metals Corp. indicated that they had reservations that 1,000 parts per million was in fact an action level and causing lead problems in the community, but they would definitely participate in any health monitoring study to help identify if there was a problem and what the extent of the problem was.

At that time NL Industries, the parties that were handling the third smelter that shut down in 1979 did not make any commitment of any kind at that time. The next critical date is Sunday, June 1. And I could relay secondhand what transpired, but I think it might be more appropriate if Ms. Phillips addressed that.

Ms. Phillips. I need to back up just a little to tell you about our meetings on the 28th and our reasons for the meetings on the 28th. On May 26 or 27, we got a call from a reporter at the Dallas Morning News, who said that he felt like he had enough information about the study, that he was intending to run a story in either the Friday or the Sunday edition of the Dallas Morning News—just the results of the study without any interpretation or without any agency plan of action, because we had not, in fact, released the report.

I was very concerned about what I determined to be a premature release of the report, and therefore I asked our Public Affairs Office to contact the reporter and ask him if he could wait until Monday, June 1, to run his story, so I could meet again with the State officials, the local officials and the companies, officially release a copy of the report and try to firm up an action plan for cleanup. That was the event, the May 26 or 27 phone call from the press, that triggered our May 28 meeting.

Our meeting on the 28th was not only to give the companies a copy of the report, but to get them to commit in writing to something that we could put in a press release relative to their oral agreements to clean up what was determined to be a problem. After we had met with the two companies on Thursday and gotten their commitments on Saturday, we met with National Lead on Sunday.

And on Sunday afternoon, after I felt like I had a package tied up in a bow, ready to release on Monday morning, I called Washington and placed a telephone call to Anne Gorsuch's office on Sunday for three purposes. One was to inform her of the potential premature release on Monday morning of the results of the study without EPA releasing that study. Second, was to assure her that EPA intended to release the results of that study that afternoon with EPA's own press release indicating what the study meant to us, and with a commitment from the State and local officials to assist us, and with a commitment from the companies to take whatever remedial actions were necessary. The third item of concern was relative to the concerns of Dixie Metals Corp., where they wanted to know if in fact we had gotten headquarters' approval of the 1,000 parts per million level that we were using as a cleanup level.

I wanted to warn Ms. Gorsuch that she may be, or her key staff people may be, receiving direct contacts from this company relative to our headquarters' commitment. So it was for the purposes of informing headquarters of my plan of action that I made the telephone call on May 31 to Washington.

Shortly after that I received a telephone call back from Dr. Hernandez. I relayed to him essentially the information we have told you: That we had done an initial study, 25 percent of the samples around the Dixie facility and around the RSR facility showed soil lead levels of 1,000 parts per million, where we were to date, and what I perceived as our commitments and plan of action.

Dr. Hernandez at that time expressed concern about the 1,000 parts per million level. He wanted to know about the literature search that we had done and what went on from a science review standpoint to determine 1,000 parts per million was the action level. He also wanted to review a copy of the press release that we were about to issue on June the 1st.

Mr. DAVIS. The only item that I think Ms. Phillips might have omitted is that Dr. Hernandez at that time indicated that before we proceeded with any cleanup level, that he recommended health studies to determine the appropriate level.

Mr. LEVITAS. Before you continue with your chronology, which is very helpful, were you aware at that time—either of you, or to your knowledge was anybody in the Agency—of some of the health situations that were existing in the communities in your targeted area of the sort described by Mrs. Spears earlier? did you have any information of sick children manifesting lead poisoning symptoms?

Mr. DAVIS. It is almost 2 years ago, and I am a little fuzzy on this. But I think there were several newspaper articles about health problems. We were obviously very, very concerned about this, and were in contact with health experts, particularly the city of Dallas. The 7,000 children that were tested between 1972 and 1979 were part of a blood screening program, and we couldn't use the data because there was only 27 within the smelter area.

We asked the Health Department if they were concerned about this and what they did when they ran into problems of elevated lead in children, let alone lead toxicity or lead poisoning. And they indicated that any time that there was a suspected problem, particularly with lead toxicity which is essentially defined as 30 micrograms per deciliter blood lead and 50 micrograms per deciliter FEP, that they had a personal follow-up with each of the parents of the children to make sure that they received adequate treatment.

Health activity at the city level particularly, but also at the State, were not overly concerned with the information either from the media or that they themselves had collected.

Mr. LEVITAS. All right, you may proceed.

Mr. DAVIS. On June 10 we asked the companies to come back in, explained that we no longer were requiring——

Mr. LEVITAS. Did you issue the press release?

Mr. DAVIS. Yes.

Ms. PHILLIPS. Yes; let me pick up from there. On Monday, June the 1st, we did Magnafax a copy of the press release to Washington. We also sent a bibliography of the studies that our staff had used to recommend to us the 1,000 parts per million level. And we received a revised version of that press release from Washington, which we issued on June the 1st.

Mr. LEVITAS. All right. Do you have a copy of the draft release and the changes that were made?

Ms. PHILLIPS. Yes, sir, I do.

Mr. LEVITAS. Could you submit the copy of both of those for the record?

Ms. PHILLIPS. Yes, sir, I will.

[The following were received from Ms. Phillips:]

EPA ENVIRONMENTAL NEWS RELEASE

The U.S. Environmental Protection Agency (EPA) Region 6 today released a preliminary study, funded by the Agency, investigating lead and cadmium levels near industrial sources in Dallas. The study shows elevated lead levels in soil near three lead and battery plants.

Samples of lead and cadmium were taken from residential neighborhoods "and schools" substitute that lie in a test area within two miles of Dixie Metals Company, NL Industries Inc., RSR Corporation and General Battery Company and within a reference area remote from known sources of lead and cadmium. Analysis of indoor dust and tap water samples revealed no significant problems with respect to lead or cadmium. Analysis of soil samples indicated that, although cadmium levels were not significantly high, lead levels in two test areas did show marked elevation in the test area above levels in the reference area.

The study shows soil lead levels in the test areas ranging from an average of 131 parts per million (ppm) to 1,253 ppm. The average soil lead level in the remote reference area was 54 ppm.

A 1978 study of lead levels in soil near major traffic arteries in Dallas indicated an average of 128 ppm.

EPA has begun meeting with State and local officials and representatives of the companies to determine the existence and the scope of any environmental problem and to discuss possible corrective actions. EPA has no evidence of medical injury to residents of the test area.

Dallas city health officials are reviewing historical blood lead information collected during the past few years through the Early Periodic Screening, Diagnosis and Treatment Program and the Children and Youth Clinic in west Dallas to determine if there is any correlation between the elevated soil lead levels and any increased blood lead levels of children residing in the study areas. No evidence, to date, of any lead concentration justifying medical intervention has been found.

However, a number of children, not necessarily living in the areas presently being studied have exhibited lead levels above what is considered normal levels.

Frances E. Phillips, Acting Regional Administrator of EPA for the five state area including Texas, says, "The voluntary commitment and assistance of the companies involved is commendable. RSR Corporation in particular took the lead in this process."

In response to the study, RSR will develop, with EPA, an expanded soil sampling program to determine the extent and magnitude of the environmental problem around its facilities, if any; and support additional biological monitoring to the extent deemed necessary. In addition, RSR has indicated it will take remedial measures as necessary to reduce the soil lead levels in high risk areas around its plant, such as playgounds, day care centers, and school yards where lead concentrations are over 1,000 ppm.

Dixie Metals Company has indicated a willingness to cooperate in corrective measures where needed, including an expanded soil sampling program and the cleanup of soil areas if necessary. They have also indicated a willingness to financially support needed additional blood sampling.

The boundaries of the two areas of further study are as follows: The first test area, located in south Dallas, within a two mile radius downwind of Dixie Metals Company and NL Industries Inc. facilities is divided into two sections. The first section is located on the north flood plain of the Trinity River bounded by Lamar on the south, I-45 on the east, AT&SF Railroad on the west and by Ervay and Forest on the north. The second section is located on the south flood plain of the Trinity River bounded by the Trinity on the north, Sargent Drive on the east, Cedar Crest Blvd. on the west and on the south by Kiest, Rector and Nolen.

The second test area, in the vicinity of RSR, located in west Dallas, is bounded by the Trinity River on the north, Norwich on the west, T&P Railroad on the south and Kingsbridge on the east.

To assist those living in the test areas who are concerned about possible lead effects, EPA and Dallas health officials advise them to contact their family doctor or Dr. Lowell Berry, Dallas City Health Department, at (214) 670-6634, if any symptoms appear. According to Dr. Norman E. Dyer, Chief of EPA's Hazardous Materials Branch, the most common symptoms of lead poisoning are anemia, severe intestinal cramps, paralysis of nerves (particularly in the arms and legs), loss of appetite and fatigue. Immediate remedies are available and individuals can be detoxified with proper medical care.

Persons wanting additional information about the study should contract Dr. Norman E. Dyer, EPA, at (214) 767-2734.

U.S. ENVIRONMENTAL PROTECTION AGENCY

Date: June 1, 1981.
Subject: Dallas Lead Study Press Release.
From: Deborah DeBerry-Kershaw, Director, Office of Public Information (60PI).
To: Byron Nelson, Press Officer (A-107).

Here's the article which appeared in this morning's Dallas Morning News. Also, to follow, is the release we've prepared as the cover press release for the Dallas Lead Study. We plan to release it at 1:00 p.m. CDT (2:00 p.m. EDT). Some minor fine turning is still in process.

Attachments.

EPA ENVIRONMENTAL NEWS RELEASE

The U.S. Environmental Protection Agency (EPA) today released an EPA funded study, investigating lead and cadmium levels near industrial sources in Dallas. The study shows elevated lead levels in soil near three lead and battery plants.

Samples of lead and cadmium were taken from residential neighborhoods and schools within two miles of Dixie Metals Company, NL Industries, RSR Corporation and General Battery Company and within a reference area remote from known sources of lead and cadmium. Analysis of indoor dust and tap water samples revealed no significant problems with respect to lead or cadmium. Analysis of soil samples indicated that, although cadmium levels were not significantly high, lead levels did show marked elevation in the test areas.

The study shows soil lead levels in the test areas ranging from an average of 131 parts per million (ppm) to 1,253 ppm. The average soil lead level in the reference area was 54 ppm. A 1978 study of lead levels in soil near major traffic arteries in Dallas indicated an average of 128 ppm.

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Frances E. Phillips, Acting Regional Administrator of EPA for the five state area including Texas, says, "We anticipate the continued cooperation of the companies, and commend RSR Corporation for taking the lead in the voluntary commitments made by the companies involved."

In response to the study, RSR will develop, with EPA, an expanded soil sampling program to determine the extent and magnitude of the environmental problem around its facilities, if any; and support additional biological monitoring to the extent deemed necessary. In addition, RSR will take remedial measures to reduce the soil lead levels in high risk areas such as playgrounds, day care centers, and school yards, where lead concentrations are over 1,000 ppm.

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Persons wanting additional information about the study should contact Dr. Norman E. Dyer, EPA, at (214) 767-2734.

Mr. LEVITAS. Identify, if you would, the changes that were made in the draft prepared at the regional office. The changes that were made in that draft as compared to the one that was revised at the headquarters.

Ms. PHILLIPS. Would you like for me to submit that or testify to it?

Mr. LEVITAS. I would like you to testify to that.

Ms. PHILLIPS. The first change related to the opening paragraph of the press release. In the draft that the region submitted, we said "the U.S. Environmental Protection Agency released the study." The Washington version said that region VI released a preliminary study funded by the Agency, so that initial change related to being more specific as to who released the study, and characterized it as a preliminary study.

The second change is in the second paragraph——

Mr. SNYDER. Excuse me, Mr. Chairman. Would it be proper, when she is discussing these changes, for her to indicate which ones that the region was in agreement with and which ones they were in disagreement with?

Mr. LEVITAS. Certainly.

Mr. SNYDER. Thank you. If you would do that. I assume the first one you wouldn't have any objection to one way or the other.

Ms. PHILLIPS. I wouldn't have any objection.

Mr. SNYDER. Thank you.

Ms. PHILLIPS. Although it—well, I wouldn't have any objection.

The second paragraph, first sentence, said, "Samples of lead and cadmium were taken from residential neighborhoods and schools within 2 miles of Dixie Metals, NL Industries, RSR Corp. and General Battery Co., and within a reference area remote from known sources of lead and cadmium." That was changed to delete the word "and schools," so it read "Samples of lead and cadmium were taken from residential neighborhoods that lie in a test area within two miles of Dixie Metals," et cetera, on to the end of the sentence.

The last sentence in that same paragraph read in the region VI version: "Analysis of soil samples indicated that although cadmium levels were not significantly high, lead levels did show marked elevation in the test areas." That was changed to add "in two test areas" after the words "lead levels did show." It was "lead levels in two test areas did show." The word "marked elevation" was dropped, and "in the test area" was changed to read "above levels in the reference area."

I think the significant things there in our view was that schools were dropped from the press release and that the adjective "marked" in "marked elevation" was dropped from the press release. And I think these two items were related to the public concern that these words might bring about.

Ms. PHILLIPS. In response to Mr. Snyder's question, I do have to tell you that Dr. Hernandez and Ms. Gorsuch had only been on the scene at the headquarter's level for approximately 15 days. Their desires and likes and approaches to problems were relatively new, not only at the headquarter's level, but at the regional level.

During this period of time I have to honestly say that our objections to changes were not very vocal at that point in time. At this point in time, not being a public relations specialist, if Dr. Hernandez or his press people said they wanted to take out "marked elevation", I took out "marked elevation", and was willing to discuss that with them.

We were doing a quick turnaround on this press release. We wanted to release it by the afternoon of June 1, and he just had it from the morning of June 1.

The next paragraph had a closing sentence that was completely eliminated at the headquarters' level. That sentence said, "A 1978 study of lead levels in soil near major traffic arteries in Dallas indicated an average of 128 parts per million."

We believed at the time that that sentence was significant because it was in essence not only a reference number, but it was relating to the fact that lead in soil was caused not only from things like smelters, but could also be caused by automobile traffic and especially arteries.

The next two changes are—the two remaining changes are on page 2. The first paragraph on the second page of our press release had a closing sentence that was completely eliminated at the Washington level.

That sentence said:

> However, a number of children, not necessarily living in the areas presently being studied, had exhibited lead concentrations above what is considered normal levels.

That was completely eliminated. That, again, was a reference sentence to us to let people know that there were other children that had lead levels above normal. I think the key to your inquiry, though, is in the next paragraph, and in the press release that was sent to Washington relative to the nature of the commitment of RSR. The sentence was in the press release that had been approved by RSR as to their commitment as follows:

> In addition, RSR will take remedial measures to reduce the soil lead levels in high risk areas such as playgrounds, day care centers and school yards where lead concentrations are over 1,000 parts per million.

That was changed to read:

In addition, RSR has indicated it will take remedial measures as necessary to reduce the soil lead levels in high risk areas around its plant.

Mr. **Levitas**. I think that is critical. I wonder if you would explain the significance of those changes.

Ms. **Phillips**. It relates to the commitment that RSR had made to the region. In my view, the press release that was sent to Washington that said RSR will take remedial measures specifically stated where they would take those remedial measures, and specifically stated the limits at which they would clean up.

The 1,000 parts per million level, and over, was the actual commitment that RSR had made. The press release that we got back was a softer version of that commitment.

It said that RSR has indicated that it would clean up, rather than that they would clean up. It had eliminated the 1,000 parts per million level that we had thought was an action level.

Mr. **Levitas**. Well, why was the 1,000 parts per million put in the region's press release? It appears to me from what you have said and also from the press release that there should be clean up of areas of lead over a 1,000 parts per million.

Was there a decision made, a policy decision made with respect to that and to the areas related to it?

Ms. **Phillips**. Dr. Hernandez was concerned about the precedent that cleaning up at an action level of over a 1,000 parts per million would set, not only in the Dallas area, but relative to all the urban areas and lead smelter areas in the country.

He was also concerned that the scientific literature may not support the 1,000 parts per million level. He preferred that we do a health study to determine the action level.

I have to say that the 1,000 parts per million level was one of many levels that were discussed as we tried to determine an action level.

So we were receiving views from everyone on what was the appropriate level.

Mr. **Levitas**. Dr. Dyer, you are a toxicologist. What is your view of the matter?

Mr. **Dyer**. Based on the literature that we reviewed during this time, I think we had sufficient evidence based on studies conducted by Dr. Carol Angle in Nebraska, Dr. Landrigen, and many others, to indicate that at least 1,000 parts per million should be an action level.

That was the reason for recommending that corrective measures be taken in those areas having a 1,000 parts per million or more.

Mr. **Snyder**. Their April 21 letter was the only thing you had in writing from them; is that correct?

Ms. **Phillips**. I had two documents in writing. I had the April 21 letter. Then on May 28, I asked the company to submit to me in writing the commitment that they could place into a press release, which they did submit in writing.

Mr. **Snyder**. Does that mention the 1,000 parts per million level?

Ms. **Phillips**. Yes, sir.

Mr. **Snyder**. OK. I was just trying to see if they had made a commitment on a thousand.

Ms. **Phillips**. Yes, sir. They had.

Mr. **Snyder**. Thank you.

Thank you, Mr. Chairman.

Ms. **Phillips**. They had approved the specific language prior to submitting it to Washington.

Mr. **Snyder**. You submitted this press release to RSR?

Ms. **Phillips**. I submitted a draft release to RSR on April 28 and asked them to give back to me not an editing of the press release, but their commitment that could be added to my press release as to what they were willing to clean up to. Yes, sir.

Mr. **Snyder**. Thank you, Mr. Chairman.

Mr. **Molinari**. Would you yield?

Mr. **Levitas**. I will be happy to yield. Congressman Molinari has a question at this point.

Mr. **Molinari**. I have a great concern as to the method in which the standard was established. What is general policy with respect to the establishment of a national standard? How does it usually come about?

Mr. Davis.

Mr. **Davis**. For most of the national standards, whether it be under the Clean Air Act, the Resource Conservation and Recovery

Act, there is a literature survey. Generally, there is inspection of facility, looking at what a facility is capable of doing and what environmental data looks like. Then a national standard is established.

Mr. MOLINARI. By whom?

Mr. DAVIS. The national program. In the Clean Air Act, the critiera document generally is done by our Office of Research and Development.

Then the Office of Air Quality Planning and Standards would evaluate the data. The standard would be promulgated as a national standard.

The situation you are dealing with here is that there are no soil standards for anything, including lead. You are essentially dealing with a problem on a case-by-case basis, particularly with lead, because the pathway is so critical and the local conditions are so critical, such as alkalinity of soil, the binding up of the lead particles, they wouldn't be mobile.

Things like this. So the agency, under no Federal statute, has a mandate to establish national standards for a pollutant in soil. The present standard that would be applicable and that the agency is essentially wrestling with, is soil contamination under the Superfund Program.

Mr. MOLINARI. Ms. Phillips.

Ms. PHILLIPS. I was just going to add that the 1,000 parts per million—and I want to make sure that we understand this because I think I have to give some deference to some of the other toxicologists' views in the regions and elsewhere—the 1,000 parts per million was an interim measure for cleanup activity while we did one study to determine if that level needed to be taken further downward.

Some people were concerned that we should clean up to the background level, which was in the reference area, which was 54 parts per million.

Other scientists in the country were saying that a 300 parts per million level was the appropriate clean up action level.

Sometimes when the agency does not have a specific standard established, and there is a concern for public health, we allow, or allowed at that time the courts to help us define that standard and give us the action level to clean up to.

We were proceeding in Federal court on some RCRA cases that were giving us some guidance in that regard. This case was different because rather than go to court and get the court to do it, I felt like I had a voluntary commitment from the company to begin that work, not in the enforcement mode.

I had no specific enforcement agreement at this time other than a commitment from the company. Not that we were not prepared to take enforcement if necessary if that commitment fell through at any point. But because the company had made a commitment to clean up, we were trying to determine what was an action level that all the scientists could agree at that point was an interim measure, and we all seemed to settle on the 1,000 parts per million.

Dr. Hernandez did not agree with that, however, after his review of the literature.

Mr. MOLINARI. Thank you very much.

Mr. Levitas. Before you proceed, isn't it true that there are no standards levels in many of these cases, and yet you still take enforcement, where the situation on that case-by-case basis warrants it?

Ms. Phillips. Yes, sir. You don't always have to have a number, either, when you are cleaning up in remedial action.

Mr. Levitas. What was the difference in this case?

Ms. Phillips. The difference in this case was that we did have some scientific literature that we were going to use. The scientific literature did give us what we believed at that time to be an appropriate action level.

We were trying to proceed as quickly as possible because of the potential health risk.

Mr. Snyder. Indeed, it was a very conservative action level.

Ms. Phillips. Yes, sir. It was very conservative in our view at that time.

Mr. Levitas. Now, because I don't want to get ahead of the story, but by the same token I don't want to get behind the story, of all of these concerns that were now being manifested about lead levels in the soil, and health problems that were being reported in the press and so forth, I just wanted to go back a bit.

On March 5, 1981, Dr. Dyer wrote a memorandum to a Bob Janney in the Office of Toxic Integration. Who is Bob Janney and what is the Office of Toxic Integration? Where is it?

Mr. Dyer. That is the headquarter's office under the Office of Pesticide and Toxic Substances as it currently exists. Bob Janney was a staff person working in the Office of Toxic Integration.

Mr. Levitas. You sent him a copy of the Dallas lead study?

Mr. Dyer. That is correct.

Mr. Levitas. In fact, you sent him some others, but you said the Dallas study should take top priority?

Mr. Dyer. Yes.

Mr. Levitas. Now, he wrote you back?

Mr. Dyer. That is correct.

Mr. Levitas. Some comments. And I just want to read them into the record. We will make the entire document part of the record.

But he said, "However, the data from the RSR area and particularly from the DMC area," Dixie Metals, I take it, "begin to cause concern."

This is Mr. Janney in headquarters, about human health, especially for susceptible groups such as pregnant women and small children. "Currently the Field Studies Branch staff uses a thousand parts per million as a warning level for lead in soil."

"This is based on the work of Yikel, et al., which demonstrated that exposure of small children to this concentration of lead in soil can result in elevated blood levels, hazard criteria are exceeded when more than ten percent of the samples in an urban area are greater than a thousand parts per million."

Then you make reference to the soil levels of the DMC and RSR area. You say those soil lead levels in the DMC and RSR areas, according to Mr. Janney's evaluation, exceed the criteria. Follow-up work should be done in these areas.

Now that information was available to you in region VI, and obviously was available at headquarters well before the end of May and early June 1981.

Mr. Dyer. That is correct.

Mr. Levitas. Was that part of the basis for your use of the 1,000 parts per million as your standard?

Ms. Phillips. Yes, it was.

Mr. Dyer. Yes, it was.

Mr. Levitas. Thank you. You see what concerns me—again, I don't understand with all of this background, with what you were finding, why the big slowdown came, and the concern about 1,000 parts per million as being a standard for doing something, rather than going back and having some more blood studies being done before you did anything.

That is my concern. In light of what we know and what the silence was telling us and what headquarters was saying, maybe you can enlighten us on that, Ms. Phillips, or Dr. Dyer.

Mr. Dyer. Yes, I would like to make a comment. We were aware of the information that Mr. Janney referred to, and several other articles, that showed the same type of soil/lead relationships.

That was the reason we were very concerned about the situation and recommended the cleanup at those levels. So, yes, I agree. And in most cases, as you have pointed out, action is based not on the actual existence of a problem, but, it is based on the potential for a problem.

But in this case we had evidence of human exposure. So we had more information in this situation than we normally have in other enforcement cases.

Mr. Levitas. You raise a very important point. Here you had—you say in most cases you take action based upon the potential for health hazard.

Mr. Dyer. Right.

Mr. Levitas. Here you had a situation where there was not only potential, based upon your soil studies, but actual evidence of exposure to the most vulnerable population, small children.

Mr. Dyer. Correct.

Mr. Levitas. And you were ready to go forward. Now, was there a shift between the end of May and the first of June in a policy of going forward where there was potential, plus actual exposure, and what came out of headquarters from Dr. Hernandez?

Ms. Phillips. I think I can respond to that. After the press release on June the 1st that had been reviewed by Washington, during that week, approximately June 3, or June 4, somewhere around that time, I did get a call from Dr. Hernandez.

He specifically told me that he had reviewed the literature; that he believed that the thousand parts per million level could not be supported in the literature; that he did not think we should spend any money to remove dirt or have any bulldozers start up until we could identify a specific health problem relative to this case.

He asked us at that time to prepare a status report and options paper to send him on the situation. I went back as a result of that telephone call and talked about the matter with my staff.

My staff was very concerned, I might add, and suggested that I might not have been forceful enough in my discussions with him about what this meant from a precedent standpoint to the region, and asked that I go back and speak with him one more time about this, which I did.

But the change, or the shift that you see here was as a result of a telephone conversation to me from Dr. Hernandez in which he specifically said that he didn't think the literature supported the thousand parts per million; that he wanted us to do a health-related study in this particular instance to show a correlation between the blood lead levels and the elevated levels in the soil.

Mr. LEVITAS. Dr. Dyer, did you think there was need for further study?

Mr. DYER. No, I did not. I don't feel that we have to, so to speak, reinvent the wheel each time we run into a pollutant problem. There are many studies in the literature done by reputable scientists all over the world, not only the United States, showing the soil/lead relationship.

So based on those studies, I felt we had sufficient information to take corrective measures.

Mr. LEVITAS. One other point in that regard. Dr. Hernandez apparently read the draft press release with respect to the commitment and undertaking by RSR to take specific cleanup actions.

Now, I don't want to characterize his testimony before our committee last week which asked him, did he call for a stop to the efforts to clean up out there. He said that he was not aware of any specific commitments by any of the smelters to take such action.

Can you enlighten me on that, Ms. Phillips? You were in direct conversation with him.

Ms. PHILLIPS. I think perhaps he forgot. It was several years ago when this happened.

Mr. LEVITAS. You think he forgot. Whether he forgot or otherwise, was he correct, based upon your knowledge?

Ms. PHILLIPS. No, sir.

Mr. LEVITAS. I am sorry. I didn't hear you.

Ms. PHILLIPS. No, sir.

Mr. LEVITAS. Thank you. Would you proceed with the chronology?

Mr. DAVIS. Mr. Chairman, before I proceed, I would like to point one thing out in fairness.

Mr. LEVITAS. Sure.

Mr. DAVIS. As Dr. Dyer indicated, the general consensus in the regional office that as an interim measure, 1,000 parts per million was appropriate, and we needed studies below that. But I would like to point out that there is extensive literature, and there are disagreements among the lead experts. The literature is not explicit that 1,000 parts per million is beyond a shadow of a doubt the lead level that causes problems; that it is not possible to develop an equation that says *x* parts per million in the soil results in Y-micrograms per deciliter in the blood.

So from a purely scientific, technical standpoint, the correlation is not there. But the consensus in the regional office, the consensus of the people that we were dealing with felt that 1,000 parts per million was the appropriate level.

But I don't want to imply that the literature was 100 percent clear that, yes, that is the right level.

Mr. **Levitas.** Dr. Davis, let me make it clear, and this is one of the real deep concerns that I have about this example of policy change. We are not talking about writing for scientific or medical journals.

We are talking about protecting the public from exposure to toxic chemicals and materials. Whether or not there is in the literature or the science a direct correlation, the question that addresses itself to a health protection agency is, do you have enough information to move at that point, or do you have to run another 18 months of blood studies before you take any action, particularly where there was already a commitment by one of the companies to go forward.

That is the question.

Mr. **Davis.** Definitely.

Mr. **Levitas.** All right.

Mr. **Davis.** But I also think that since the time of June 1981, at least two extensive studies were done. We now have the, quote, definitive report in. And when we get into the final report and what the recommendations are, what does the final recommendation from CDC and the peer review panel recommend? Is it 1,000 parts per million cleanup?

A correlation? I am sure we will get to that, but I want you to keep that in mind, also.

Mr. **Levitas.** We will get to that, of course, but the question, of course, that I am asking and that others are asking, and certainly the parents of the children in the targeted areas have been asking is, could you have reduced their exposure to lead had action been taken in June 1981, rather than waiting for 18 months to do so?

Would you have reduced their exposure to this lead toxic condition?

That, I think, is the question that we have to ask.

Mr. **Davis.** Well, let me reassure you that the management and professionals in region VI, and I am sure in headquarters, were very concerned about that very question. To this day, looking back, should we have done it differently?

I am not sure of the answer.

Mr. **Levitas.** All right. Dr. Rowland is trying to get recognition. Before I do, Dr. Dyer, would you like to contribute anything to this point?

Mr. **Dyer.** Well, I think in regard to the soil lead/blood lead relationships, as Dr. Davis indicated, there is quite a bit of variation in terms of some of the reports. But I think the preponderance of information, recent studies, clearly shows that there is a soil/lead relationship.

When you have soil levels of 1,000 parts per million or more, it doesn't take a lot of the soil being ingested by the child to elevate the blood lead levels. Just putting hands in the soil every day and then putting those dirty hands in his mouth, or the toys in his mouth, can elevate the blood lead levels, because you are talking over 1,000 parts per million of lead in the soil, and only 0.3 part per million of lead in the blood to cause the elevation.

So when you look at those two, it is very possible for that soil to raise the child's blood lead level above the 30 to a dangerous level.

Mr. **Levitas**. Are you aware of any standards elsewhere in the world that were different from the 1,000 parts per million as being an action level?

Mr. **Dyer**. Many countries have some levels that they have used for various purposes. Toronto has used, I believe, either 500 parts per million or 600, something in that range, for some cleanup efforts.

The State of California in cleaning up an area for a park used the—I believe it was 500 or 600 parts per million level. I forget exactly which.

But they based that on the Consumer Product Safety Commission's standard for paint, which limits the amount of lead in paint to about, well, it bans lead in paint above those levels, above 500 to 600 parts per million.

The State of California used that 500 to 600 parts per million level in park cleanup some years ago. There are some others around the country.

Mr. **Levitas**. Thank you. Dr. Rowland, you wanted to ask a question?

Mr. **Rowland**. Yes. Thank you, Mr. Chairman.

Dr. Davis, Dr. Dyer, also, would you say what might be a toxic level for one individual in the soil or wherever may not be toxic for another individual?

Mr. **Dyer**. It is all dependent on exposure. Yes; the person's nutrition certainly would affect whether or not the level would be toxic. As you know, the diet that a person consumes, how nutritious it is, what minerals are there, some minerals actually impede the absorption of lead from the intestine.

But in areas where you have a large number of poor people that might not be getting the highly nutritious diets, you could have a greater absorption of the lead from the gastrointestinal tract than you would from a person who had a very highly nutritious diet.

So there are some differences there. But then, again, it depends on the frequency that the individual is exposed. Many studies have shown differences between boys and girls living in the same area.

The reason is that little boys get out and get dirty and put their dirty hands in their mouth more frequently than girls.

So there are many differences.

Mr. **Rowland**. I guess the point I am trying to make is that 1,000 parts per million may be toxic for one individual whereas it might not be toxic for another individual; would you say that?

Mr. **Dyer**. No; I think if a person was exposed to that in the same manner, I think the person would absorb enough of that lead to cause blood-lead elevations.

Now, it is a matter of exposure. Just living in the area would not necessarily mean that they are exposed to——

Mr. **Rowland**. And the variation in individuals themselves could make the difference.

Mr. **Dyer**. Yes, variation in individuals themselves.

Mr. **Rowland**. So it might be very difficult to set a standard?

Mr. **Dyer**. As far as soil lead is concerned?

I think any standard should certainly look at the use of the land, the use of the particular contaminated area. If the area is used as a playground for kids, then certainly we should be as—we should select a number to provide as much protection for those kids as we possibly can.

Because as we all know, we get lead from other sources, and the body can handle a certain amount of lead, excrete it as you take it. But then you reach a point where the body cannot excrete it fast enough.

So for small kids playing on school playgrounds or parks or residential play areas, I think we should look at the maximum protection.

Mr. ROWLAND. But then we should look more at the individual, we should look more at the children, maybe, to see what their health situation is. That might be more significant than trying to say a certain lead level in the soil or whatever is the important thing?

Mr. DYER. Well, what we have done, and what the literature has done, is looked at them. Looked at the soil/lead relationship in children in certain areas, recognizing that there is some exposure from air, there is some exposure from foods, and then the exposure from the soil is an additive effect that adults normally would not have.

So I think the studies have taken that into consideration. The efforts that we were proposing have taken that into consideration.

Mr. ANDREWS. Would the gentleman yield for a question?

Mr. ROWLAND. Yes, I will yield.

Mr. ANDREWS. In truth, Doctor, aren't the lead contents six to seven times higher in children than they are in adults?

Mr. DYER. In normal—normally you are talking about?

It depends on where the child resides. If you would look at the Johnson Study conducted in Dallas, the traffic study conducted in Dallas, it showed children in that area with concentrations, well, below 12.

Mr. ANDREWS. But six or seven times more dangerous than adults; isn't that correct?

Mr. DYER. Oh, more times dangerous in children than in adults?

Mr. ANDREWS. Yes?

Mr. DYER. Yes.

Mr. ANDREWS. Yes?

Mr. DYER. Because you are looking at developmental systems, systems in development in children, whereas in adults, most of those systems are already matured.

Mr. ANDREWS. Were those factors that were considered in 1981 and 1982 within your agency as to what to do and what your judgment was on whether or not the EPA should clean up that area?

Mr. DYER. Of course.

Mr. ANDREWS. Is that correct, Mr. Davis?

Mr. DAVIS. Yes. Going back to an earlier question, the primary indicator of a health problem obviously is the health effect. In this case we were using the CDC guidelines for lead poisoning in young children.

Their definition of lead toxicity is 30 micrograms per deciliter of blood lead, 50 FEP. Now, the reaction to that situation is it is definitely a warning situation, and you want to identify the pathway that the children are getting the lead from, and reduce or remove that pathway.

There is an entire spectrum from that alert level of lead toxicity through symptoms and ultimately, lead poisoning, which is essentially defined by CDC, I believe, as 80 micrograms per deciliter and a significantly elevated FEP.

So when you are in a situation of 30 blood lead, 50 FEP, you really have potential health problems and that is the area that we are very concerned about. So we focused in on that as essentially our action level and we were looking for environmental contamination that would produce that, or higher levels.

Mr. LEVITAS. Thank you. Yes, Mr. Whittington.

Mr. WHITTINGTON. Mr. Chairman, I would like to make one point. I arrived on the scene in this affair in late September of 1981. Within about a week of the time I arrived, I did schedule an appointment with Dr. Hernandez to go to Washington and request that he form a committee of people to look at the question of what constitutes a problem of lead in soil, in other words, trying to arrive at an action level. He did appoint such a committee.

There is such a report. They concluded that you could not properly set one, and I will provide that report to the committee if you don't already have it.

Mr. LEVITAS. We are familiar with the report in the committee, and we probably will have some questions about it later on. We would appreciate it if you would provide a copy of that report.

Mr. WHITTINGTON. Yes, sir.

[The information received from Mr. Whittington follows:]

A REPORT ON A STUDY TO DETERMINE THE BLOOD LEAD LEVELS IN DALLAS CHILDREN

A pilot study of blood lead levels in Dallas children was done in July 1972, by members and staff of the Dallas City Health Department, and the Dallas Area Respiratory Health Association in cooperation with volunteers from two participating areas in the city. The study had two aims:

1. To determine the range of blood lead levels in Dallas children who live in the two areas tested.
2. To determine whether residence near a point source of lead emissions to the atmosphere has any influence on blood lead levels in Dallas children.

STUDY DESIGN

The test area, referred to as to the "Cadillac addition", is bounded by Morrell, Cedar, and Sargent street, and the M.K.T. railroad. Two secondary lead smelters, Dixie Industries and National Lead, are located on the eastern border of the Test Area. Prevailing winds during much of the year are south easterly. Extensive data on the lead emissions of these smelters have been accumulated by the Air Pollution Control Division of the Dallas Health Department. The racial composition of the area is approximately 75% Mexican American and 25% black. Most homes are single dwellings approximatey 20 years old.

Two control areas were selected since no single area with similar housing, socioeconomic level, and racial composition could be identified. One area, bound by Canada Drive, Brantley, Pueblo, and Pastor was predominantly Mexican American; the other, bounded by McBroom, Morris, Vilbig, Shaw, and Puget was predominantly black. Both areas closely resemble the Test Area in age of homes and socioeconomic level.

TABLE 2.—LEAD ANALYSES

	Test Area	Control area
Blood lead(mcg/100 ml):[1]		
Number of samples	27	26
Mean	30	22

TABLE 2.—LEAD ANALYSES—Continued

	Test Area	Control area
Standard deviation	7	8
Standard error	1	2
Soil lead (ppm):[2]		
Number of samples	14	15
Mean	727	255
Standard deviation	451	206
Standard error	120	53
Ambient Air Lead (mcg/100 ml):[3]		
Number of samples	8	4
Mean	3.5	1.5
Standard deviation	1.8	.1
Standard error	.7	.1
House paint lead (average of 5 sides per house mg/cm^2)[4]		
Number of samples	15	14
Mean	.68	1.0
Standard deviation	.54	.55
Standard error	.14	.15

[1] $t=3.816$ $p<.01$ (by Wilcoxon Matched Pairs Signed—Ranks Test, $p<.01$).
[2] $t=3.57$ $p<01$.
[3] $t=3.08$ $p<02$.
[4] $t=1.61$ $p<05$.

All occupied homes in each area were given a number; homes were then selected for study by reference to a random numbers table. Selected homes were visited by neighborhood volunteer workers who determined whether or not children aged 2-6 years lived in the home, and whether the parents would consent to the drawing of a blood sample from the child. A future date was established for blood drawing and signed parental consent obtained.

Five ml. blood samples were drawn in vacutainer tubes containing anticoagulant, and were airmailed to United Medical Laboratories in Portland, Oregon, for lead analysis. Prior to receipt of the results, study subjects from the Test and Control Areas were paired by age and race, in cognizance of the findings of others that blood levels are age-dependent and higher in blacks. Results were analyzed by parametric and non-parametric statistical techniques.

Studies in other metropolitan areas have related elevated blood lead values in small children to the lead content of the paint in the home. Peeling paint, found especially in dilapidated housing is ingested by the children. Accordingly, the lead content of the paint on four inside walls and one outside wall was determined in each home in which the family participated in the study, using a radioisotope fluorescent analyzer borrowed from the Department of Health, Education, and Welfare, Bureau of Community Environmental Management. Readings from the five sites were averaged to obtain a single value for each home.

Samples of ambient air in both Control Areas and the Test Area were collected with hi-volume samplers by the Air Pollution Control Division and the samples obtained were analyzed for lead by their usual technique.

Samples of soil, approximately one cubic inch, were collected from the front yard of each participating family. Sites were chosen to avoid obvious contamination by vehicular emissions. The samples were analyzed by the Air Pollution Control Division.

RESULTS

Over half of the eligible families participated in the study (Table 1).

TABLE 1.—FAMILIES PARTICIPATING IN STUDY

	Eligible	Participating	Percent
Control area:			
Black	12	7	58
Mexican-American	14	12	86

TABLE 1.—FAMILIES PARTICIPATING IN STUDY—Continued

	Eligible	Participating	Percent
Total	26	19	73
Test area:			
Black	8	4	50
Mexican-American	20	11	55
Total	28	15	54

Results of lead analyses are shown in Table 2. A complete listing of the data is included as Appendix 1. The frequency distribution of blood lead values is shown in Figure 1.

COMMENT

These data indicate that 5 of the 53 (9%) blood samples equaled or exceeded 40 mcg/100ml, a level generally accepted as a potentially harmful level.[1] These high levels were equally distributed between Test and Control Areas but could not be related to the lead content of household paint. However, as a group, the blood lead levels of children in the Test Area were 36% higher than that of children in the Control Areas. This difference was statistically significant by both parametric and non-parametric techniques. The Environmental impact of the two lead smelters was shown by the increase in both air and soil lead contents in the Test Areas.

The long-range biological significance of these findings is unknown. However, in view of the known effects of lead associated with blood levels only slightly higher than those reported here it is clear that further studies are required.

RECOMMENDATIONS

1. A larger study of children should be done to evaluate lead exposure in various areas of Dallas.
2. Means now exist to pinpoint sources of environmental lead, based on the differentiation between lead isotopes by mass spectrometry. Such a study has been proposed by the University of Texas at Dallas. We recommend that this proposal be implemented and made part of the recommended study of lead exposure in Dallas.
3. The need for regulations to restrict the emission of lead to the atmosphere is confirmed by this study; it is therefore recommended that enforceable regulations to that end be adopted.

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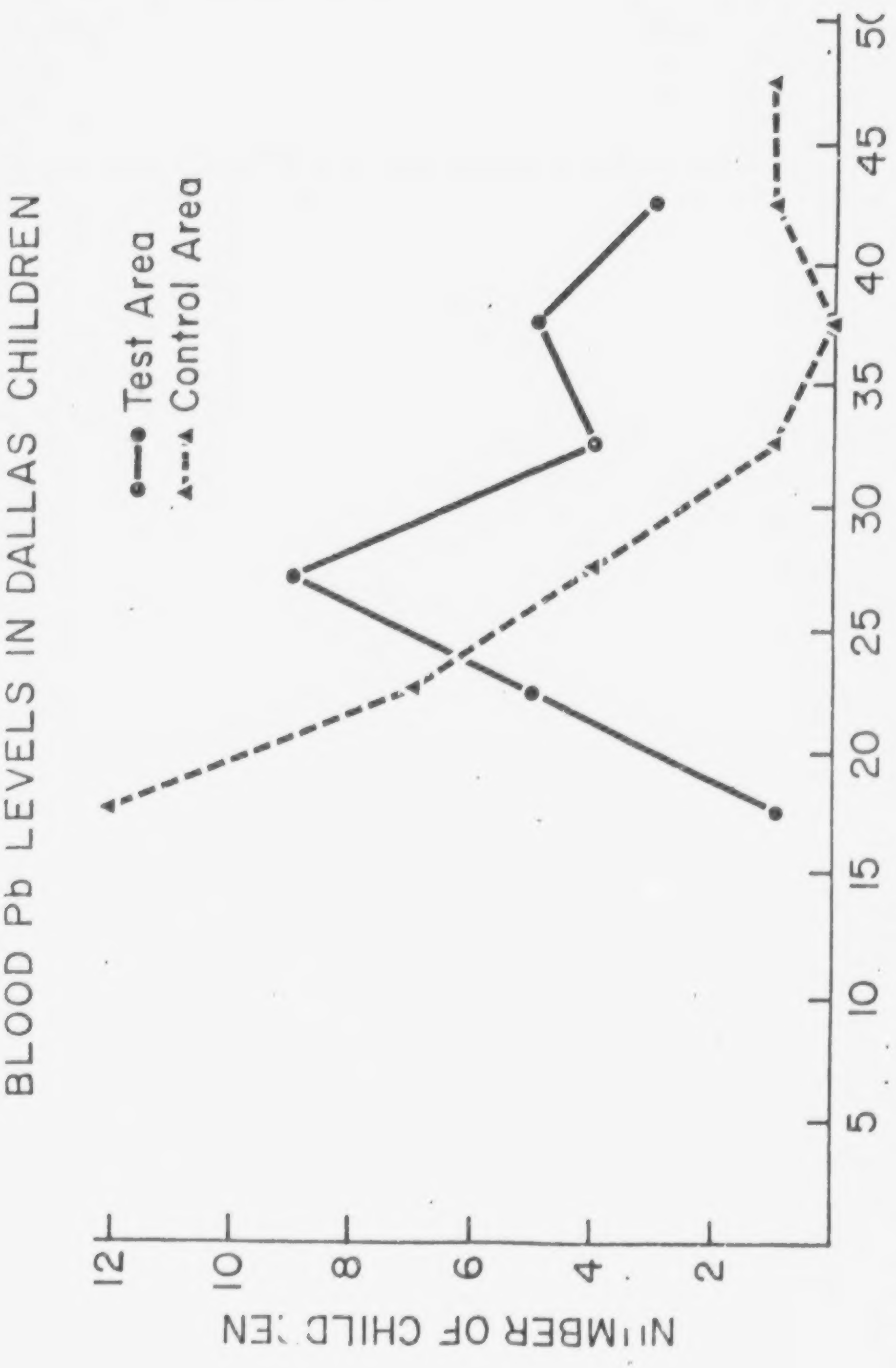
[1] Medical aspects of poisoning H.S.H.M.A. Health Report Vol. 86 P. 140 1971.



BLOOD Pb LEVELS IN DALLAS CHILDREN

RESULTS OF BLOOD LEAD ANALYSIS

T–c	Test area		Test results meg/100 ml	Control area		Test results meg/100 ml
	Age	Ethnic group		Age	Ethnic group	
18	2	Mexican American	35	2	Mexican American	17
15	2	do		2	do	28
−9	2	do	31	2	do	
19	2	do	38	2	do	19
4	3	do	28	3	do	24
18	3	do	34	3	do	16
−2	3	do	24	3	do	26
−3	3	do	25	3	do	28
4	3	do	27	3	do	23
18	4	do	38	4	do	20
16	4	do	32	4	do	16
6	4	do	27	4	do	21
11	4	do	29	4	do	18
14	5	do	30	5	do	16
13	5	do	26	5	do	13
5	5	do	24	5	do	19
0	5	do	16	5	do	16
7	6	do	20	6	do	13
−2	6	do	26	6	do	28
7	6	do	22	6	do	15
−4	2	Black	27	2	Black	31
−18	2	do	27	2	do	
−18	2	do	38	2	do	
16	5	do	36	5	do	20
1	5	do	24	5	do	23
21	6	do		6	do	19
16	6	do		6	do	24

Note.—Wilcoxon Matched—Pairs, Signed—Ranks Test Level of Significance for two-tailed test p 0.01

Mr. LEVITAS. Then if you want to continue the chronology, I want to get back to what my concern is. My concern is that the information on the lead in the blood, the lead in the soil information, the literature—it appears to me that the regional office of EPA had made a decision to go forward and clean these areas up and that that got off-track as a result of the intervention by headquarters EPA; and that that decision to clean up, and spare these people to exposure to lead levels of the magnitude we were talking about was stopped, and further studies were called for.

That is what I am concerned about.

Mr. SHAW. Mr. Chairman, could I ask a question?

Mr. LEVITAS. Absolutely.

Mr. SHAW. Mr. Davis, in your testimony a few minutes ago you made reference to the fact that it wasn't known beyond a shadow of a doubt. Now if that is the level of proof that is being required, then I would suggest that we had better reconsider this standard.

I think that when Dr. Dyer talked about the preponderance of evidence, that that had better be the rule of thumb and what we had better work with, because when we are talking about human health and the health of young children, pregnant women, and people who are extremely vulnerable, we cannot use a test such as beyond a shadow of a doubt. That is not acceptable.

Perhaps, Mr. Chairman, this is where the difficulty lies. Perhaps this is what the test difference is. Perhaps this is what we really should be zeroing in on and working on.

Mr. LEVITAS. Thank you. I think you have stated it very well.

Mr. MOLINARI. Mr. Chairman, we have talked a lot about the situation and I think we have gone around in a circle without hitting the question head on. Is it the opinion of the officials from region VI that Dr. Hernandez interfered with the cleanup of the site by RSR and others?

Mr. DAVIS. I would say yes.

Mr. MOLINARI. Ms. Phillips.

Ms. PHILLIPS. He changed the direction that we were going, No. 1. And—he did change the direction that we were going, without a doubt, yes, sir.

Mr. MOLINARI. I want to go a little further than that. Did he interfere with the cleanup process that was contemplated by region VI?

Ms. PHILLIPS. Yes, sir, he interfered. I do want to add one thing to that, however. The company, RSR, in particular, had made a commitment to the region to clean up, as an interim measure, the 1,000 parts per million level and over. Even though EPA changed its position on holding them to that commitment, RSR did go out and clean up, to my knowledge, most of the areas that had 1,000 parts per million level or over despite our not requiring them to honor their commitment.

Mr. MOLINARI. Now, I am a little confused.

Ms. PHILLIPS. OK.

Mr. MOLINARI. I am hearing from one person that he interfered with the cleanup and I am hearing from another member of the panel that indeed they went ahead with the cleanup nevertheless. The question is did he exert his influence to prevent a cleanup from taking place?

Mr. DAVIS. I would say he changed the direction of the regional office's strategy. That one of the companies voluntarily went out during the summer of 1981 and essentially followed through with our preliminary recommendations is to the company's credit. But by changing direction, the regional office was not going to require that, and if the companies refused to clean up at the 1,000 parts per million level, there was always the potential of litigation in our back pocket.

Mr. MOLINARI. All right. Go ahead, Ms. Phillips.

Ms. PHILLIPS. I was just pausing to swallow hard.

Mr. MOLINARI. My concern is this. Did Dr. Hernandez's role create a health hazard that would not exist today, or perpetuate a health hazard which would not exist today?

Ms. PHILLIPS. I don't think that we could—that I could deny that, if we had gone forward in June and picked up 1,000 parts per million, both companies at that point in time, we would have reduced the potential exposure of those children during the interim in which we studied the matter further.

Mr. MOLINARI. So putting it conversely, had Dr. Hernandez not injected himself into this matter, the health risk to the children in the area would be less today than it would have been had he not interfered? Is that a fair statement?

Mr. DAVIS. It is a very hard question to answer. Let me jump ahead now until the conclusion of both the city's survey and then the final EPA-CDC survey. We come out with a final report in January—in February 1983. Now, I am jumping ahead a year and a half. In some areas we were wrong. It turned out that the ambient air around the company was, at least during the summer and fall months, twice the national ambient air quality standard. We had assumed that there was not a serious air pollution problem around RSR, we assumed that there was not an air problem around either facility, and our calculations based on modeling indicated to us if there was an air problem, and it was questionable, we thought it would be around Dixie. So we were fairly surprised after we started monitoring. The Texas Air Control Board and the city started monitoring in April 1982. The soil levels at both Dixie and RSR, even though at RSR, the soil contamination was a little higher and a little further out, generally they were approximately the same.

Dixie did not show lead toxicity in the children tested. RSR showed 5.6 percent of the children with lead toxicity.

Mr. **Molinari**. How does that compare to the national average for urban areas?

Mr. **Davis**. At RSR, I think it is below the national average. Obviously, the Dixie situation with no lead toxicity is below the national average. There were elevated levels of lead in the blood above normal, normal being a reference area for Dallas. But if the soil levels are the same at both facilities, and the major difference is air, the indication is that air would be the principal reason for the lead toxicity in the children.

There is a definite contribution of the soil, and the recommendation of CDC and the peer group panel was to address the soil within a half mile around both smelters, not just RSR, where there was clearly a lead toxicity problem, but also Dixie, but they were concerned about any increase in blood lead around the facilities due to undue exposure of the children. So their recommendation addressed day care centers, schools, playgrounds, public use areas, things like that.

So it is very difficult for me to answer your question, did the change in direction on June 1 have a health impact on the community.

Mr. **Molinari**. Let me turn the question over to Dr. Dyer. Since you are a toxicologist, Dr. Dyer, I think you are probably the one on the panel that would be best suited to answer that question. Would you mind giving us your thoughts on the subject?

Mr. **Dyer**. On the turning of the decision and its impact on the health of the kids?

Mr. **Molinari**. Exactly.

Mr. **Dyer**. I would say definitely, because you have to consider, as I said earlier, all routes of exposure. Now, if there is air pollution, we were essentially told that the air emissions were in compliance with the regulations at that time——

Mr. **Molinari**. Who told you that?

Mr. **Dyer**. We were advised that by the State and the air program, based on whatever data they had. OK. So not knowing that the air emissions were in violation, we said OK, the kids are still being exposed to lead from some source. Now we must also consider that there were elevated blood lead levels not only in the RSR area, however. There were also elevated blood lead levels in the Dixie area as a result of the Dallas screening effort, so we just can't throw those out at this point, because they had to get their lead from somewhere.

Mr. Molinari. Let me try a shortcut if I can.

I am trying to zero in on this particular problem and how it might have impacted the children that reside in the area. Dr. Hernandez changed the direction of region VI as they were going to approach the problem. As a consequence, the cleanup that was contemplated may or may not have taken place. What I am trying to get to is the decision of Dr. Hernandez. Dr. Dyer, did you say his action definitely had an impact upon the health of the children?

Mr. Dyer. OK, I had no interaction with Dr. Hernandez.

Mr. Molinari. I didn't ask that. I just want to know in your professional opinion whether it had an adverse health impact on the children in the area.

Mr. Dyer. Let me say that the change in the decision, and the decision not to proceed with corrective measures definitely had an impact on the health of the children.

Mr. Molinari. Then what did you do as a toxicologist from region VI? Did you express your strong disapproval, or did you contact Dr. Hernandez and say, "You are making a tragic mistake"? I would like to know what you did.

Mr. Dyer. I expressed my disapproval.

Mr. Molinari. How did you do that? In what form?

Mr. Dyer. Verbally in the regional office and, well, later on, and I guess through this committee, through the national committee that Dr. Hernandez eventually appointed.

Mr. Molinari. Did you not deem this serious enough to reduce your objection to writing so that somebody couldn't say you didn't call or send a letter to the Washington headquarters, saying we have a serious problem on our hands?

Mr. Dyer. I did that eventually.

Mr. Molinari. When did you do that?

Mr. Dyer. I initially—OK. The exact date on my initial correspondence I think was after the Dallas blood screening data had been reported showing that I believe 13 percent of the kids residing within 0.5 mile of the facility had blood lead levels over 30. And then at that point when there was a call, still, for additional study, and criticisms raised against the Dallas screening effort and the soil data regarding quality assurance and self-selection as far as the blood screening, I wrote a memo and distributed it to the task group that was appointed by Dr. Hernandez, voicing my strong concern about what it was doing, and indicating that I think—I felt there was sufficient data available to show that there was a problem, and that the contribution from the traffic was minimal, based on other studies, not just this one, but several other studies conducted by Dallas.

Mr. Andrews. May I have leave, please?

Mr. Molinari. Sure.

Mr. Andrews. Do we have a copy of that memo?

Mr. Dyer. Yes, I think you do.

Mr. Andrews. Dr. Dyer, one question. Have you kept notes and memorandums throughout this investigation?

Mr. Dyer. The investigation? Oh, the—you mean the soil studies?

Mr. Andrews. Yes.

Mr. **Dyer**. I have kept some records of communications, yes.

Mr. **Andrews**. Have you ever been asked by anyone or instructed by anyone not to make notes of your actions or memorandums of your conclusions or recommendations?

Mr. **Dyer**. Instructed——

Mr. **Andrews**. I mean that question in the very broadest context, Dr. Dyer. Has anyone ever told you not to make notes, or encouraged you not to keep personal records of your conversations or activities, or any other matter relating to not just this committee's investigation, but of the continuing chronological events that were happening in Dallas? Anyone told you not to keep notes?

Mr. **Dyer**. I don't—OK. I don't recall any specific instructions regarding keeping notes. I recall once when we, just prior, or just after an FOI, freedom of information request, there was concern about the notes that were available and this type of thing, that had opinions rather than factual information.

Mr. **Andrews**. Expand on that, please. What do you mean?

Mr. **Dyer**. Well, notes in which individuals were expressing their opinions regarding such—regarding situations. And as such, those opinions were obtained by the freedom of information seekers, and were published in some papers——

Mr. **Andrews**. Including your own opinions?

Mr. **Dyer**. Yes; that was including my opinion. There was concern about those being written down. But I did not receive any specific instructions that I recall saying not to write anything.

Mr. **Andrews**. Who had the concern about your opinions being written down?

Mr. **Dyer**. OK, this would have been, well, it would have been my superiors.

Mr. **Andrews**. How did they express that concern to you about your opinions being written down?

Mr. **Dyer**. I don't recall the specifics on that. But I suppose—there were instances in which we would get into arguments regarding statements that I had made or actions that I had taken.

Mr. **Andrews**. Were you instructed not to write your opinions or conclusions down?

Mr. **Dyer**. I don't recall ever receiving specific instructions with regard to that, no.

Mr. **Andrews**. Do you know whether or not anyone in the Dallas agency was instructed or told or encouraged not to write their opinions down on paper?

Mr. **Dyer**. I think probably based on the conversations that I was talking about, that some individuals might have received that——

Mr. **Andrews**. What individuals?

Mr. **Dyer**. I can't say, all of them, who would be involved. But——

Mr. **Andrews**. Tell us who you know.

Mr. **Dyer**. But I can't name any specifics because I don't recall the specifics at this time. I am just thinking in terms of the general, the general framework, the general attitude, the general atmosphere that existed at that time. I don't recall the specifics on conversations or instructions.

Mr. **Levitas**. Thank you. The gentleman from New York.

Mr. **Molinari.** I would like to pick up where I was before. You had some concerns as a result of the holdup or the intervention of Dr. Hernandez. Did you communicate your concerns to Ms. Phillips, who was then acting regional administrator?

Mr. **Dyer.** I communicated them definitely to Dr. Davis who is my superior, and I am not sure now whether I talked directly to Ms. Phillips, but I am sure that the message regarding my concerns did reach Ms. Phillips, yes.

Mr. **Molinari.** Ms. Phillips, who did you hear from within the region with respect to the concerns about the holdup here in Washington.

Ms. **Phillips.** The two main people that I heard from relative to the concerns were the two people that I expressed the change of direction to, that was Dr. Davis and Diana Dutton, our enforcement division director. When I expressed my—when I expressed what Dr. Hernandez had said relative to the fact that we were going to do a health study as opposed to continuing with the commitment that we had made, I was in a meeting with some of the enforcement division staff. Ms. Dutton was director of the enforcement division.

At that point in time, not only Dr. Davis and his staff, but the enforcement division staff, as I mentioned earlier, were very concerned that perhaps I had not been forceful enough in expressing to Dr. Hernandez what this was going to mean to us from an implementation standpoint. I asked the key staff to prepare some speaking points for me, or to discuss with me some speaking points relative to this change of direction so that I could go back to Dr. Hernandez and bring this up again.

Mr. **Molinari.** Did they prepare those speaking points?

Ms. **Phillips.** Yes; they did.

Mr. **Molinari.** Do you have a copy of them with you?

Ms. **Phillips.** Unfortunately I do not. I have tried to refresh my memory on some of the main items that were in those speaking points. I think it was a matter of sitting down, saying let's talk about it; what do I need to tell Dr. Hernandez and making a list of those things to go back to him with.

Mr. **Molinari.** Let me interrupt you. Is it that you don't have them with you, or that you can't locate them? Do you have them back at the regional office?

Ms. **Phillips.** I have not searched for them in the file.

Mr. **Molinari.** May I ask that you search for them and, if you find them, furnish a copy for the subcommittee?

Ms. **Phillips.** I would be glad to.

Mr. **Molinari.** But I do understand that there was disappointment on the part of some of your colleagues that you weren't forceful enough in bringing home the point to Washington headquarters?

Ms. **Phillips.** Yes, sir.

Mr. **Molinari.** What is your reaction to that? Did you believe that you were forceful enough?

Ms. **Phillips.** I thought perhaps I wasn't, and I called Dr. Hernandez back to go over the points that had been brought up by the staff. We were working at the time on a very short time fuse, if you will recall. And Dr. Hernandez gave me direction and said this is what he wanted us to do, and I said, yes, sir, that was the direction that we would go.

Mr. MOLINARI. Was this in the nature of an order?

Ms. PHILLIPS. This was in the nature of a telephone conversation.

Mr. MOLINARI. But didn't he say this is the way we are going to go and that is it?

Ms. PHILLIPS. It was an order to me, yes, sir.

Mr. MOLINARI. It was an order.

Ms. PHILLIPS. I called Dr. Hernandez back after I had gathered the speaking points together and went over, as I recall, from my best recollection, four major points. One of them was a primary concern for the children. I reminded him that this was in a poor area of Dallas, and the region's concerns for the children. Second, I talked to him about, again, what we have been discussing here, what was perceived to be a change of direction at that time in our operations that we had gone from action on a potential health problem to having to prove an actual health problem before we did anything. And I specifically talked about that in relation to the effect this had on present negotiations in other Federal court cases in the region where we were using section 7003 of RCRA and other statutes, and how this could affect our negotiations and how, last, this could affect our future decisionmaking.

Mr. MOLINARI. All right.

Ms. PHILLIPS. Those were my main points.

Mr. MOLINARI. Did you ever learn why RSR went ahead voluntarily to clean up? Did they communicate their reasons why they did proceed even though there was a holdup on whether they should or not from headquarters?

Ms. PHILLIPS. I think that they had made a corporate commitment and——

Mr. MOLINARI. I understand that, and they are probably to be congratulated for making that commitment. But since there was a holdup and they decided to go ahead, it seems to me that they must have had a judgment that it was the prudent thing to do. I am just wondering if you have any information to share with us as to how and why they made that decision.

Ms. PHILLIPS. Well, when we initially set the 1,000 parts per million as our regional action level, we reviewed with them the literature that we used to come up with that number as a——

Mr. MOLINARI. By them, you mean RSR?

Ms. PHILLIPS. RSR, yes, sir.

Ms. PHILLIPS. And I think that they believed that it was the prudent thing to do.

Mr. LEVITAS. Thank you. I would like to recognize the gentleman from Kentucky at this time. Then we will go around again, because the significance of this change and shift in policy is of deep concern to me, and its implications.

The gentleman from Kentucky.

Mr. SNYDER. Thank you, Mr. Chairman.

Ms. Phillips, you wrote a memorandum to Dr. Hernandez on July 7, 1981, recommending cleanup by the companies. That is correct, isn't it.

Ms. PHILLIPS. July 7?

Mr. SNYDER. Yes, ma'am.

Ms. **Phillips**. OK.

Mr. **Snyder**. Attached to that was a matrix chart showing actions to be taken for various blood lead levels and various lead soil concentrations. You are familiar with that?

Ms. **Phillips**. Yes, sir.

Mr. **Snyder**. That chart recommends for blood lead levels above 30 and high lead soils greater than 1,000 parts per million, that site cleanup was recommended: Is that correct?

Ms. **Phillips**. Yes, sir.

Mr. **Snyder**. That chart recommends the same cleanup where blood lead levels were above 30 and lead in soils at medium levels, or 500 parts per million. Is that correct?

Ms. **Phillips**. I didn't hear the second half of it, I am sorry.

Mr. **Snyder**. The chart recommends the same cleanup action for sites where the blood lead levels were above 30 and lead in soils at medium levels; that is, above 500 parts per million?

Ms. **Phillips**. Yes.

Mr. **Snyder**. I am leading up to a point that Mr. Davis made in response to a question by Mr. Molinari. On what date did you have sufficient information to make specific cleanup decisions using these criteria for each of the three smelter impacted areas; that is, both adequate blood lead and soil data?

Ms. **Phillips**. OK. The chart that you are referring to is in response to Dr. Hernandez's request for an options paper—that an options paper be prepared and sent to him that would indicate what the region's actions would have been if we found various elevations of blood lead. That is what that chart is related to. And Dr. Davis, what did we use——

Mr. **Snyder**. My question was——

Ms. **Phillips**. I am sorry.

Mr. **Snyder**. On what date did you have sufficient information to make the specific cleanup decisions using the criteria for each of those three smelter impacted areas?

Mr. **Davis**. The definitive answer was provided in the CDC peer group final report——

Mr. **Snyder**. CDC. That's the Center for Disease Control?

Mr. **Davis**. Yes, sir.

Mr. **Snyder**. That came in February of 1983?

Mr. **Davis**. Yes, sir. What I would like to point out, though, the left-hand column is 30 micrograms per 100 milliliters or greater. That is elevated blood lead. That was taken from the CDC report. As you see, in that column every action is site cleanup. So it didn't make any difference what the soil lead levels were, if that was the soil lead level that produced the 30 micrograms per deciliter we were going to clean up.

Then the second column is 20 to 30, which would be increased blood lead over normal, but still below the definition of CDC's action level CDC's definition for lead toxicity. Then the far right-hand column is less than 20 micrograms, which essentially would be anything in the generally normal range. In the middle area we were concerned, but we were going to go with the public education program because the CDC action level hadn't been exceeded.

The left-hand column is when the CDC action level had been exceeded, and then no matter what the soil was we were going to take action, soil cleanup. So the answer to your question is we really never used the criteria until CDC gave us the interpretation of the data.

Mr. **SNYDER**. When you made those recommendations for a cleanup of the areas where the blood lead levels were above 30, soil levels above 500 ppm, that was based on the belief that RSR's smelter was actually in full compliance with its lead air levels. Is that correct?

Mr. **DAVIS**. Yes.

Mr. **SNYDER**. Was RSR in compliance with the EPA regulations under the Clean Air Act for smelter lead emissions in June of 1981?

Mr. **DAVIS**. The data that we have is the data starting April 7, 1982. We have three quarters of data. Two of those quarters, the spring and the summer quarters, showed excedances of the national ambient air standards. The third quarter showed they were in compliance. But you are essentially talking about the monitoring closest to the RSR smelter. So they were exceeding the national air quality standard but they were not in violation of any State or federally enforceable emission limitation. In other words, the regulation that applies to the smelter.

Mr. **SNYDER**. When was noncompliance established, if it was?

Mr. **DAVIS**. That the smelter was violating, was exceeding, the national ambient air quality standard——

Mr. **SNYDER**. The EPA regulations under the Clean Air Act.

Mr. **DAVIS**. Yes. Standards. When we received the data from the Texas Air Control Board in the fall of 1982.

Mr. **SNYDER**. So the noncompliance really was established by the Texas Air Control Board?

Mr. **DAVIS**. It was a joint monitoring network between the city and the State, partially funded by EPA.

Mr. **SNYDER**. Could the high blood lead levels found in 1981 and 1982 in children living near the RSR smelter be caused by air emission occurring at that time from RSR facilities?

Mr. **DAVIS**. It could have, yes.

Mr. **SNYDER**. If the change in direction had not been made, which has been the matter of some discussion and controversy here, would one of the principal causes to wit, air emissions, have been determined in an orderly fashion?

Mr. **DAVIS**. Yes, we were working with the Texas Air Control Board to resolve the questionable modeling around both facilities. And it was just a question of when the Texas Air Control Board, or the Texas Air Control Board requiring the companies to conduct ambient monitoring around the smelters would start.

Mr. **SNYDER**. What have you determined are the primary and secondary causes of sources of lead in blood for the residents living around there?

Mr. **DAVIS**. The final report indicated that the contribution from the smelter was on the order, ratios, 5.5 from the smelter to 1 for the traffic.

Mr. **SNYDER**. That determination was just made in January or February this year?

Mr. **DAVIS**. Yes.

Mr. SNYDER. Before that report in January or February of this year, had the EPA ever had a large-scale technical study that established a supportable link between the soil lead levels and blood lead levels?

Mr. DAVIS. Not that I am aware of.

Mr. DYER. There is some indication of that in the clean air standard for lead. There is a discussion that there is a link in that standard.

Mr. SNYDER. Dr. Davis, you indicated that RSR exceeded the national ambient air quality standards. Did they have a permit of some kind?

Mr. DAVIS. They have a permit from the Texas Air Control Board. At that time I believe the Texas Air Control Board also had emissions limitations for lead. EPA had not approved those yet because of the controversy over the acceptability of the model——

Mr. SNYDER. Were they in compliance with their permit?

Mr. DAVIS. As far as I know they were in compliance with their State permit.

Mr. SNYDER. If you had taken cleanup action on those hot spots in 1981 where the lead soil levels were over 1,000 ppm based on that 1981 study, would the total areas be the same as that found in the January 1983 report?

In other words, did we learn anything new about soil contamination by the fact that direction of the cleanup action was changed?

Mr. DAVIS. The original EPA study in October–December 1980 by the University of Texas at Arlington did for us, and then the subsequent followup environmental sampling that the city did generally identified some hot spots, but it wasn't very extensive. When EPA finally went in and did the study in the summer of 1982, we did extensive environmental monitoring, particularly for soil, and very accurately was able to plot the soil isopleths. So the recent study identified the extent of lead in the soil around both facilities.

Mr. SNYDER. In attachment A to your January 1983 or February, whichever is proper, study, the statement appears:

> The previous blood lead screening program by the city of Dallas resulted in an inability to reach firm conclusions. The recommendation was made to do a study of a valid sample of preschool population.

Is that consistent with the last answer you just gave, or is my unscientific mind tying the wrong things together?

Mr. DAVIS. I think it is consistent, and the study I was referring to was the study that resulted in that conclusion. In other words, that——

Mr. SNYDER. That conclusion was only arrived at this year?

Mr. DAVIS. Right. In February.

Mr. SNYDER. When was the recommendation made with respect to a valid sample?

Mr. DAVIS. That came out of the results from CDC and peer group review of the city study of October, November, December 1981. CDC-EPA came out with its evaluation of the study in April of 1982 that recommended a more definitive study by the Federal agencies.

Mr. SNYDER. Did CDC ever indicate during this period of time that an imminent health hazard existed?

Mr. DAVIS. Not to my knowledge.

Mr. SNYDER. Did anybody on your staff indicate an imminent health hazard existed or there was a prosecutable case?

Mr. DAVIS. I would say various members of the staff, including Dr. Dyer, expressed their concern that they felt there was an immediate health hazard. However, the medical people that we were dealing with in EPA, Washington, and subsequently with CDC and the peer review, in both the review of the Dallas report and also in the final report, indicated that there was no indication of lead poisoning. The data that was available had indicated no probability, possibility in their mind for that happening. But you did have lead toxicity, which obviously is of concern.

But I don't think that CDC or the peer group review in either report gave any indication of their feeling of a medical health problem.

Mr. SNYDER. I have been listening a whole lot today. I haven't asked a whole lot of questions until right now. It appears to me that there is perhaps an overriding allegation that Dr. Hernandez's scientific judgments were in error. That seems to be the drift of what I get from the testimony that I have heard thus far.

I want to ask all four members of the panel, aside from that, do any of you have any evidence that there is any, or has been any unholy alliance between Dr. Hernandez and any of the three companies that we have mentioned this morning?

Ms. PHILLIPS. No evidence.

Mr. WHITTINGTON. I don't, sir.

Mr. DYER. I do not.

Mr. DAVIS. I don't.

Mr. SNYDER. Do you have any reason to believe that any actions on his part were based upon anything other than his scientific judgment, even though it may have been in your opinion in error?

Mr. WHITTINGTON. I do not.

Ms. PHILLIPS. I do not.

Mr. DAVIS. No.

Mr. DYER. No.

Mr. SNYDER. Thank you, Mr. Chairman.

Mr. LEVITAS. Thank you, Mr. Snyder.

Just to make the record clear on that point, though——

Mr. SNYDER. Does the chairman want to respond to the question?

Mr. LEVITAS. Yes. In case there is any, in case you want to go beyond that, I am not simply questioning Dr. Hernandez's scientific judgment, I am questioning and challenging his policy judgment and his enforcement judgment, and the change in policy that he brought about as a result of this decision.

Mr. SNYDER. I think all of that has been placed in question. Perhaps what I just said in the area of his scientific judgment was not all encompassing. But to get to the reason for that, I asked the latter two questions. Apparently they believe it was error in judgment on his part in all of the areas that the chairman has mentioned, but not based upon anything corrupt between him and any of the three companies. That is the essence I get from the testimony.

Mr. LEVITAS. And I will just say I am not aware of any corruption. I am aware of a policy, as I view it, by Dr. Hernandez and the

Agency, a change of policy that placed children health at risk due to exposure.

Mr. SNYDER. Just pursuing the discussion a little bit further, Mr. Chairman, in some of our previous hearings there has been some testimony that might indicate some unholy alliances between some people at EPA and some companies.

Mr. LEVITAS. Oh, yes, there is no doubt about that.

Mr. SNYDER. But I have not yet heard that allegation with regard to Dr. Hernandez. I think if it is to be made, it ought to be made for the record. If it is not there, then there ought not to be an inference of it. That is why I asked the question directly.

Mr. LEVITAS. Thank you. And I appreciate your making that point. I will say this. I know the gentleman has another engagement. But it is my intention at some point to ask Mr. McAnalley, who is a toxicologist from Dallas and formerly with the Agency, to join us on the panel with Dr. Dyer and express their views on the CDC and the 1983 study, peer review, to further discussion along, because I think it is important to get into that. I will be glad to discuss it with you later in the day.

Thank you.

Mr. SNYDER. Thank you, Mr. Chairman.

Mr. LEVITAS. Dr. Rowland?

Mr. ROWLAND. Thank you, Mr. Chairman.

Dr. Dyer, I have some questions that I wish to ask you. How long have you lived in the Dallas area?

Mr. DYER. I have lived there since 1971.

Mr. ROWLAND. 1971. Have you been a toxicologist during this period of time?

Mr. DYER. Yes.

Mr. ROWLAND. I have a paper here, a report, May 6, 1982, the subject was "Lead Study Group," that you wrote, I believe. It was directed to the lead study group liaison members. You are familiar with the paper that I refer to?

Mr. DYER. Yes, sir.

Mr. ROWLAND. In 1979 there were some studies done on lead levels of children in the Dallas area. Did these studies reveal that the lead levels may have been higher in children that lived in the vicinity of the smelters than in other areas of Dallas? That is, were there more sick children from lead poisoning in the areas of the smelters?

Mr. DYER. OK. I am not aware of the clinical history of the children or the clinical aspects of it. I don't know whether there were more sick children. I think the thing that we were initially concerned about is that in reviewing the city of Dallas blood lead screening records, there were indications of kids residing in certain areas having undue lead exposure. This is based on the lead concentration of their blood.

Now, those children, there were many problems with that data. Followup screening would show that the next time sampled the bloods were normal again. So this raised questions in our mind about that data, and we wanted to know why that was occurring. What was happening. The study I am referring to in the memo is the screening effort that took place in, I guess it would have been 1981.

Mr. ROWLAND. 1979?

Mr. DYER. 1981? OK. The screening that was completed by the city of Dallas where they screened over 11,000 children and adults showed 13 percent of the kids residing within a half mile of the facility as having elevated blood lead levels. That was certainly, to me, reason to suspect or to conclude that there was a relationship between living in those areas near the smelters, Dixie and RSR, I forget the—I think that percent was a combined percent for both areas. But looking at that data it certainly drew in my mind the conclusion that there was a problem.

Mr. ROWLAND. In your professional opinion, then, people living in the vicinity of these smelters were having more difficulty with elevated blood levels than other areas of Dallas; is that correct?

Mr. DYER. That is correct.

Mr. ROWLAND. You made a statement in this paper. You said: "As a public health agency it is our responsibility to protect the health of the citizens from environmental insult, preferably before harm has occurred," et cetera. This is what I was attempting to establish earlier when I was pursuing a line of questioning in this area about it varies from person to person. You say the Agency has procrastinated long enough so that it is now time to move with all deliberate speed to have the appropriate study and institute appropriate measures.

Since the report what have you done to do what you indicated should be done?

Mr. DYER. As far as that report, that memo, as I say, it was written after the screening results. The panel, well, we worked with the panel in developing a protocol for the additional study. The additional study has been conducted and completed, and the report on that additional study was released in February, February 1.

Mr. ROWLAND. Of what year?

Mr. DYER. Of 1983.

Mr. ROWLAND. All right. It was just that you did these studies—did you say or do anything in addition to that to try to resolve what you perceived as being a very bad situation?

Mr. DYER. Other than discussing it within the Agency, as you recall there were peer review people from outside the Agency looking at the report as well. And so based on the information that EPA was getting, the peer reviewers, I disagreed with that, but I didn't see any way I could really impact that.

Mr. ROWLAND. Do you feel the people in Dallas and EPA should have been more vigorous in doing something to correct the situation?

Mr. DYER. Yes.

Mr. ROWLAND. Why wasn't something more vigorous done then?

Mr. DYER. Because I suppose we—the comments and the opinions made by the peer reviewers and the headquarters' people tended to raise some doubts about, you know, whether or not the information available supported us taking action then. I never agreed with that, but I didn't see—I didn't see how I could impact it.

Mr. ROWLAND. That was a general feeling in the Agency, as I understand, that perhaps you may not have been right?

Mr. DYER. No, I never believed that we were wrong. I just didn't know how to change that decision, other than going ahead with the additional study, and I felt certain that the additional study would show that there was a problem, that problems still existed.

Mr. ROWLAND. Thank you, Mr. Chairman.

Mr. LEVITAS. Thank you, Dr. Rowland.

Let me go with a few questions. Then I will get back to the gentleman from New York. Dr. Davis, are you an SES, or schedule C?

Mr. DAVIS. SES.

Mr. LEVITAS. Ms. Phillips.

Ms. PHILLIPS. SES.

Mr. LEVITAS. Dr. Dyer.

Mr. DYER. No. Just general service.

Mr. LEVITAS. Mr. Whittington?

Mr. WHITTINGTON. I am schedule C.

Mr. LEVITAS. Have you worked previously with CDC in doing any health studies, Dr. Davis?

Mr. DAVIS. Prior to the lead situation?

Mr. LEVITAS. Yes.

Mr. DAVIS. I don't think so.

Mr. LEVITAS. I am very familiar with CDC. It happens to be located in my congressional district and it is an outstanding public health agency in the United States. But I have never found that in terms of health evaluations that that is their long suit. They primarily focus on rather large numbers of epidemiological studies rather than universes, particular clinical health evaluations.

I was wondering whether or not you had any discussions with CDC concerning that aspect, the large universe needed for an appropriate epidemiological study, or the smaller universe involving the children in the Dallas area.

Mr. DAVIS. The study that CDC reviewed the first time was the city of Dallas' study.

Mr. LEVITAS. Right.

Mr. DAVIS. The review by CDC and the peer group was arranged by headquarters, as was most of the contact with CDC on the final study that EPA did essentially in consultation with CDC. But as far as I know CDC expressed no reluctance to get very much involved in the Dallas situation and in fact had two or three people on site——

Mr. LEVITAS. I am not suggesting any reluctance. What I am trying to find out is whether you had any discussions with them concerning what I consider thereby as in favor of rather large universes for their evaluations.

Mr. DAVIS. No.

Mr. LEVITAS. Ms. Phillips, did the policy decision and change in policy that emanated from this Dallas lead matter, did that have any effect in any other either pending or developed case enforcement in the Dallas region?

Ms. PHILLIPS. To my knowledge, it did not. At the time, however, we were of course very concerned that it would. But we kept on in the direction we had been going in the past.

Mr. LEVITAS. Other than Dr. Hernandez, did you have any discussions with officials at headquarters, EPA, concerning this matter?

Ms. PHILLIPS. Yes, sir. I talked to some representatives in the Office of Toxic Substances. I am sure at one time or another I talked to, in that group, Walt Koralick, Joe Carra, Dr. Marilyn

Bracken. I also talked to the enforcement side of the house because our strategy was if we could not get a quick voluntary cleanup that we were always going to use our enforcement arsenal to effectuate some environmental cleanup. And we also talked to the enforcement side of the house.

I recall specifically discussions with Lamar Miller, who was head of the Hazardous Waste Task Force at the time.

Mr. LEVITAS. What were the opinions and reactions of Dr. Bracken and Lamar Miller, do you recall?

Ms. PHILLIPS. I think there was a concern in the headquarters offices that if this was not a change in direction, it was certainly a shift in the level of assurance that we had relative to some of our enforcement actions.

Mr. LEVITAS. That was a view shared both by Dr. Bracken and Dr. Miller?

Ms. PHILLIPS. Yes, sir.

Mr. LEVITAS. Is Dr. McAnalley here?

Mr. McANALLEY. I am here.

Mr. LEVITAS. Would you come up, because my next question would be put to you and to Dr. Dyer. If you would, I will swear you in while they are getting a name plate for you there.

[Witness sworn.]

TESTIMONY OF BILL McANALLEY, Ph.D., TOXICOLOGIST, DALLAS, TX

Mr. LEVITAS. If you will have a seat at the table there and give us your name, your present occupation and what your relationship was with region VI of EPA before your present career.

Mr. McANALLEY. I am Dr. Bill McAnalley. My training, briefly, is I have a degree in math and chemistry. I taught chemistry and math. I was trained on an NSF fellowship in natural science. Then I was trained under a National Institute of Health grant to produce toxicologists because of the imminent need for toxicologists. One of the bases, or one of the supports for the grant was the fact that EPA needed people who could look at chemicals and evaluate their health effects. I was trained there at Southwestern Medical School, and after that I did an internship in clinical and forensic toxicology, at which time just as I was finishing up, Dr. Garriott, whom I was training under, and he will be an important point, if I could bring him in.

He was the first person in the nation to get a doctorate in toxicology, and he is the one who wrote part of the training grant I was trained under at Southwestern Medical School. His doctorate in toxicology was received at—under an NIH grant because Lilly, or the pharmaceutical companies, the chemical companies were saying, "Hey, we are producing chemicals that help but they also have adverse effects. We need to understand something about that." And so he was trained jointly at Lilly and Indiana Medical School.

So he was contacted by the local EPA office because they wanted a toxicologist to look at their studies to see whether or not there were any problems. So Dr. Garriott, the other reason he was contacted, at that time he was the national head of—for the Society of

Toxicologists in looking for people, toxicologists looking for jobs. So I was given the opportunity to work with the EPA as their regional expert in toxicology. And at that time we had seven or eight hazardous waste sites. That was before the Eberhard list when we had several hundred hazardous waste sites but I would look at the sites specifically to determine if there was an imminent and substantial hazard. If there was, then we were to take action. That was my main function.

Mr. LEVITAS. During what period of time did your work at the EPA region extend over?

Mr. MCANALLEY. They contacted me in 1978 to see about getting a job, and I wasn't hired until 1979 because I had to come out of the national register, which I still don't really understand. But experts have to come in Washington in toxicology. They put all of your grades and courses in a computer. Then if you are a veteran you get 10 points added. If you are a disabled veteran you get 15 points added. Then everyone in the Nation gets to compete for the job. I understood there were hundreds. Pat Hudson said, "You probably won't get it." I don't know. I never got to see it. But supposedly I came back No. 1. So they had, I got the job.

Mr. LEVITAS. What period of time was this?

Mr. MCANALLEY. That was in 1979.

Mr. LEVITAS. Do you still do work with Dallas region?

Mr. MCANALLEY. No. I left the EPA about a year ago.

Mr. LEVITAS. So from 1979 to 1981 or 1982 you worked at EPA?

Mr. MCANALLEY. Yes.

Mr. LEVITAS. You said that part of your responsibility and the reason you were brought in was to ascertain and make determinations as to whether there were imminent health hazards resulting from certain toxic materials?

Mr. MCANALLEY. Yes. That is correct.

Mr. LEVITAS. Now, did you do any work with Dr. Dyer?

Mr. MCANALLEY. I did a little work with Dr. Dyer. Dr. Dyer was working in the toxic substances, and I was working with RCRA. And the work I did with Dr. Dyer is, I represented the enforcement representative to this eventual lead study that we are talking about. And Dr. Dyer and I planned most of the lead study.

Mr. LEVITAS. And so you were working on the enforcement side. And part of the purpose of your expertise was to decide whether the hazard was of such a nature that it should require enforcement action. Is that correct?

Mr. MCANALLEY. That is correct.

Mr. LEVITAS. All right. Now, would you comment on the type of basis for taking enforcement action of the sort that was in place prior to late May, early June, of 1981, and contrast that to the new policy which EPA adopted at Dr. Hernandez's recommendation?

Mr. MCANALLEY. OK. Could I give some examples of specific cases? The first case I reviewed, when we started we had seven cases. One of them was the Vertac Chemical site in Arkansas, at which time I noticed the dioxin, 2,3,7,8-tetrachlortaldioxin, or now the most toxic known man-made carcinogen toxic molecule. At that time that was not agreed upon. In fact, the first thing I did at the region when I got there, they said, "We are glad you got here. We want to know about dioxin." Bill Hathaway—Fran, you were there—everybody was talking about dioxin. And it was very interesting because the statement that they asked me to answer on or to respond to was: is dioxin toxic to people or just animals?

And that is interesting, you know, as a scientist, that they would ask is it toxic just to animals, because you see, as a general rule, like there is a 10-to-1 rule or so of rats. And that means that whatever it takes to kill a rat, or injure a rat, a lot of times it takes one-tenth depending on how the chemical works. So in pharmacology that is part of your job. So I was amazed because that came from a Washington toxicologist. That at that time Washington thought that dioxin was toxic to animals and not people. And also they thought at that time that dioxin was degraded so fast that there wouldn't be more at the site.

I said, "Why don't we check and make sure?" They checked the site. Sure enough there was still some there. I said, "Well, let's check the fish." They contacted me a little later and they couldn't catch any fish around the site. I said, "That is probably because they are dead. Go down until you catch some." Sure enough a few miles down they caught some. We found dioxin 120 miles down the stream.

We prepared our case and I was deposed on that case, at which time I emphasized the rat part so I could bring that out in testimony. And I also emphasized the fish livers because, see, dioxin is concentrated in fish livers. We had at that time looked at the catfish in the—had FDA look at it in that area because I figured if they brought out fish livers, we would say what——

Mr. McAnalley. We fingerprinted dioxin. My training in forensic science, you know, when you have a hit and run and they find a red chip and they say it comes from this car, well, what you do is you do a chemical profile of the paint.

Well, each one of the dioxin manufacturers had their own process. There is more than one TCDD. There are 22 TCDD's. We had Dr. Bob Harris at Research Triangle Park look at those different TCDD's.

So we could counter the fact that the other side was saying how do you know it came from that plant. See, well, because if it was from Bell, it had only the one. If it was from that plant, it had this profile.

We found that profile in the fish. So that way we were able to prove that the dioxin came from that plant. In fact, I made the statement if you gave me some on the moon, I could tell you who made it.

I still think we can. An interesting point there is we had one attorney who had never tried a case before, several other attorneys came in and got sick or left. The whole case was run with one attorney, Ronny Alcar, who didn't know any chemistry, and the other side had Ph.D. chemists with law degrees, you know, questioning us.

They hit us really hard on the fact that dioxin is degraded in such a short time that there is no need in testing it. We wanted them to go back again and test those same areas.

They said that we were wasting our time. Bruce Elliott finally got permission to go back and test it. The dioxin was still there.

We were criticized for spending our money, EPA's money, to go back and prove that the dioxin was still there out of Washington. So that was an interesting point about that case.

But I didn't think anything about it at that time. I just figured it was bureaucracy.

The next case that I worked on, or one before that, I was reading—in fact, this was the first imminent, substantial hazard case in, as far as I know, in the Nation under RCRA. That was at the 2001 chemical site where Louisiana said someone was making an old insecticide, or miticide, specifically to kill mites, and they made it in a sugar plantation. I mean a sugar-type factory that had an old acid container where they put sulfuric acid in this container and right underneath it were several drums of cyanide. Hundreds of pounds.

When the inspector was there it was leaking. Well, that had broken and dropped on those drums; that was an imminent hazard because there were people around there.

Mr. LEVITAS. Let me ask you this, Dr. McAnalley, because what I am trying to get at, trying to find out is based on your experience, one of the earliest toxicologists testifying in these early cases, how did the level of proof that was required for you to testify and bring these enforcement actions in these cases compare to the type of evidence of health hazard that existed in the lead sites in Dallas?

Mr. MCANALLEY. Well, in every case, all you had to have was a potential. In other words, if things were left alone and you didn't do something about it, there was a good probability that it might hurt human health or the environment.

Mr. LEVITAS. That was the basis of the enforcement.

Mr. MCANALLEY. That was the basis of every case.

Mr. LEVITAS. Was that, in your professional opinion, a legitimate basis for taking enforcement action?

Mr. MCANALLEY. Yes; we had been. We had won every case.

Mr. LEVITAS. All right. Now, are you familiar with the type of information and evidence that was available in connection with the Dallas lead site situation?

Mr. MCANALLEY. Yes; I am very familiar with it.

Mr. LEVITAS. How would you compare the quantity of evidence of potential health hazard in that case as to some of these others you have dealt with?

Mr. MCANALLEY. We had more evidence in this case than any case I had ever worked on, plus this was not an imminent hazard. It was a hazard. Because, see, in some sites we didn't even have anybody there.

Like the Midas site was abandoned. But if someone came by, it could injure them. In the Petro-processors case, as soon as they put a fence around it to keep kids off of it, you know, then the imminence was gone.

But, here, you would have to move the people off. So we had a definite imminent and substantial hazard and I said that from the very first time I saw the data.

Mr. LEVITAS. In the other cases you are talking about, there were actual cases brought and litigation held, and successfully brought to conclusion in enforcement actions. In this case, where there was more evidence than you had ever seen before, there was no enforcement action brought?

Mr. McAnalley. No; the action was started. We were starting the action. In all the other cases, we would, you know, prepare everything.

Go in and say, this is an imminent and substantial hazard, present it to the judge. Then the judge would, say, rule on it. There would be action immediately.

In fact, in the cyanide thing, I was ordered by the judge to tell them how to fix it. All they had to do was drain the acid out.

Mr. Levitas. Now, in this case, what happened? In the lead case.

Mr. McAnalley. Nothing.

Mr. Levitas. One last question I want to ask you. Then I will recognize my good friend and colleague. Are you familiar with the Lead Smelter Study Group Office of Toxic Administration Report of the Dallas Area Lead Assessment Study dated October 1, 1983? Are you familiar with that?

Mr. McAnalley. No. I am not. Dr. Dyer just told me about it.

Mr. Levitas. Dr. Dyer, are you familiar with it?

Mr. Dyer. Yes, I am.

Mr. Levitas. All right. I would like, if you would, and Dr. McAnalley, based on what you hear in this conversation or what you have been told concerning this report, you can certainly contribute. What I would like, Dr. Dyer, is if you would describe the areas or aspects of that study that you would find in any way questionable.

Mr. Dyer. OK. There are several areas. First of all, the study, the protocol states that an effort would be made to determine the blood lead/soil relationship. In other words, to try to identify the percent of kids residing in the highly contaminated areas with elevated blood lead levels as compared to the control area. That step was never taken.

Mr. Levitas. Wasn't that the purpose of the whole study?

Mr. Dyer. That was the purpose of the whole study. But that step was never taken. The other problems I have with the study is that it, some of the conclusions, it jumps to the conclusion that the only difference between the Dixie area and RSR area is the fact that there were air violations in the RSR area and not in the Dixie area, when, in fact, there are some other things also that were different.

The type of vegetation cover, or the type of cover in the areas were different, Dixie having more vegetation than the RSR area.

The time of sampling. The sampling was initiated in the Dixie area. Just prior to the sampling being initiated, there were very heavy rains throughout the month of June and the first part of July which means you had very wet conditions for at least a month prior to the sampling in the Dixie area.

By the time the sampling reached the RSR area, we had the hot, dry, normal weather underway. Those are some concerns about some of the conclusions.

Another, and I think very basic one, is the comparison of the results in the 1983 report with the Hanes II study, using the Hanes II study as a national standard, when, in fact, it does not represent a national standard.

First of all, the authors in the study caution against the use of the data for comparative purposes because of possible bias. They state that less than—well, at least 40 percent of the targeted population did not participate. So it was not a totally representative sample. They indicate in that study that they are following up on that particular aspect of it.

I guess, second, in regard to the Hanes II study, there are problems in trying to determine what the exposure of the people in the Hanes II study actually was.

We have no information on the Hanes II study, so all of those or many of those that participated probably had an interest because of some concern about lead, and so we have no knowledge whether they lived around localized exposures or not.

So to try to compare the results in that Hanes II study with the Dallas lead study, and say the Dallas results are less than the national average is inappropriate, especially in light of the fact that the Dallas study included a statistically designed control area, and in selecting the control area, an area was selected that had very similar variables in terms of population ratios, other things of this nature.

The main difference would be that the control area was located away from a localized source other than traffic. So the ideal comparison would have been, only comparing the test areas with the control area, not bringing in the Hanes II study as a comparative matter.

Mr. LEVITAS. Thank you. Now, Ms. Phillips, is it correct that this report, this study, was done as a consequence of the policy of Dr. Hernandez and EPA in order to provide the basis for enforcement action?

Ms. PHILLIPS. Yes, sir.

Mr. LEVITAS. Now, and as I recall, Dr. Hernandez is quoted as saying that we need to know what relationship is between soil lead levels and blood lead levels. Until we knew that, we ought to hold off.

Ms. PHILLIPS. Yes, sir.

Mr. LEVITAS. Now, do I understand you, Dr. Dyer, as saying that that question is not even answered by this report?

Mr. DYER. Yes. That is correct.

Mr. LEVITAS. All right. Now, that is 18 months later. We don't even have the answer.

Mr. DYER. Right. We have the data. See, we have a very comprehensive soil, geostatistical analysis which was conducted by our Las Vegas research facility. We have the blood level data.

But the two were never brought together in a final analysis to determine what that relationship is.

Mr. LEVITAS. My question to Dr. McAnalley in light of what you have heard, my first question is, in your opinion as a professional who has worked in this area, particularly in the enforcement end of it, was it, would it have been necessary to get this information about the level between soil lead and blood lead in order to bring about an enforcement action, and would you have waited to get this information before so doing?

Mr. MCANALLEY. No. I would not have waited. And, in fact, I agree with Dr. Dyer on everything. The important point that has never been addressed is that we knew—we had previous studies, soil studies, that told us what levels were too high.

We said a thousand is for sure too high for children to play on, especially with no grass. We also had a 1972 study done by the city in these two areas which told us the blood relationship or gave us an idea in the exact same sites.

Now, that is very important, because we knew how the soil responded. Let me address that.

In the studies in El Paso where it is so dusty, when you dust your house one day you can't tell in an hour you dusted it, there, very low levels can be hazardous.

They looked at the increase in soil lead concentration and plotted the increase in blood. So what you can do is relate the increase in soil with blood.

That was also done in the Silver Valley Study. It was done in Canada. It was done in numerous studies by Carol Angle. There is a direct mathematical relationship.

Mr. LEVITAS. What would have been the reason for wanting to wait until you get that information before you take enforcement action?

Mr. MCANALLEY. Well, the problem with this study, No. 1, I went to CDC's seminar——

Mr. LEVITAS. My question is why would you have wanted to wait?

Mr. MCANALLEY. There would be no reason to wait.

Mr. LEVITAS. Go ahead.

Mr. MCANALLEY. If you wait, this type of follow-up study is worthless. Let me explain why it is worthless. I am going to use CDS's own criteria. In the New Orleans seminar, Dr. Hawk was there, Dr. Naddleman, several lead experts were talking to the States, in that area, Tennessee, a huge area, and they said that when you suspect lead poisoning in children, go immediately and sample. Because what happens is when the parents know that there might be a problem, the blood leads start to fall.

If you wait too long, when you go back to sample, they will appear to be normal, or not at a problem. I told that to Dr. Davis. I told that to Fran. I told that to everyone I could tell it to.

But we waited. The other key point is we had top people in the Nation right there in Dallas. We had a pediatric neurologist, because this is something you want to know, how is it affecting their neurologic system.

The medical school was ready to go in and sample, take blood samples, once we knew the soil levels were confirmed. In other words, if they were a thousand for sure, we had to go right then.

The first thing we had to do was tell the parents, send out a letter and say, hey, there is a risk and we are going to be coming in to sample. This would be done, since the University of Texas is the richest university in the Nation. It would have been done for virtually nothing.

The contract money was already there. Right? We had Dr. Garriott who is on the Board that certifies toxicologists nationally looking over the study and it was designed, ready to go.

What we wanted to do was look in the wedge. We knew where the high levels were. Let's look in the wedge. We didn't need background. Dr. Garriott ran every child's blood level right there in his lab.

That was the charity lab they ran. In terms of background, we had them all coming in through there. The normal backgrounds in certain areas were less than 10 microgram percent.

When you have a child that comes in with 40, 50, 60, it is coming from somewhere. So what we wanted to do was get in right quick, sample the wedge. If they have problems, start treating, start chelating them because what happens with children is their blood brain barrier develops with age, and if—whenever that lead gets up in there and seals off, it is there.

So, had we found poisoned children and the study that was done by the city we are talking about here in 1972, I would like to read just a point of this. This is a pilot study of blood lead in Dallas children. It was done in July 1972 by a member of the staff of the Dallas City Health Department and the Dallas Area Respiratory Health Association in cooperation with many volunteers. The volunteers, that is one of the top medical schools in the country. So they had all the support they needed.

The conclusion of that study I think has been overlooked several times. It is signed recommendations, a large number of children should be done to evaluate lead exposure in various areas of Dallas. Means now exist to pinpoint sources of environmental lead and it says the need for regulations to restrict the emissions of lead to the atmosphere is confirmed by this study. It is therefore recommended that enforceable regulations be adopted.

This is 1972, signed by Dr. Libby and the other members of the committee.

Mr. **Levitas**. May we have a copy of that for the record?

Mr. **McAnalley**. Yes, you can. A key value here is we had one mean—the mean blood lead level around the RSR smelter was 30 microgram percent. That is the average. That means the average kid based on today's standard, the average blood level would be one of alert. The soil level, the mean soil level in that study around RSR was far less than a thousand. I have it exactly here.

Do you know what it is? Here it is.

OK. The soil mean was 727 in the test area, parts per million. In the control area, 255 is what it was.

Now what amazed me when I looked at that, knowing what the average blood level should be, if the control blood level was 22 and their soil level was 255 in the control, there is something wrong or there was really a problem there because the control levels around Dallas for these children should be 10 or less. But what happened when we started mapping these is we found that the control that the city used in this test was around the other smelters. So then it made sense because that is why in the—the soil levels were so much higher.

Outside of Dallas, like around—away from possible lead emissions, you find soil around 10. The national soil concentration is around 10 to 20 parts per million. So here we had a known slope for Dallas. In other words, we knew about how the blood and soil related, and I was thinking that whenever our data came back it would be less—hoping it would be less than 700, because 700 indicated a problem before, so if it comes back at 1,000, there is no question that is a problem.

What we told Dave is, and Fran, is that 1,000 for sure—how much lower we go, you know, we will have to see as the blood data comes in to the medical school, to Dr. Susan Nan, the pediatric neurologist, to Dr. James Kurt, one of 50 certified clinical toxicologists in the Nation at that time, to Dr. James Garriott, the person who certifies toxicologists—he calls several lead experts all over the country. They all agreed with the concern. To all of those people that was an imminent hazard. He has testified on imminent hazards.

Mr. LEVITAS. Thank you, Dr. McAnalley. I think the point is made.

Then the date of that study is 1972?

Mr. MCANALLEY. Yes.

Mr. LEVITAS. We would like a copy of that for the record.

Mr. Molinari?

Mr. MOLINARI. There are some areas of confusion in my own mind as a result of what we have heard here today. I would like to try to clear some of them up if we could. For example, I have heard that we failed to prosecute when we had a very good case. Yet we were told, on the other hand, that in the case of RSR, despite the fact there was a hold put on it, they went out and started to clean up the site nevertheless.

Mr. Davis, you heard what Mr. McAnalley said. Do you have any areas of disagreement with his testimony?

Mr. DAVIS. I am not a lead or medical expert, and the regional office in this situation was depending on the advice and counsel we were receiving from CDC on the interpretation of the lead data. Obviously there appears to be quite a bit of difference of opinion between the CDC reports, both on the Dallas lead study that the city conducted and the final CDC designed study and report; but I feel inadequate in responding to those, and I think it would only be fair to provide an opportunity to CDC and members of the peer group panel that did the work.

Mr. MOLINARI. Fair enough. Throughout the reports, the attachments and the task force report, reference is constantly made to the samples of black children as opposed to other than black. There is no explanation in here as to why the difference. I would assume that the blood chemistry has some bearing and that perhaps there is a greater susceptibility among the black population and other than black.

Can someone explain why the differentiation in the data?

Mr. DYER. In the situations in the data we have looked at, I am not sure it is clear whether there is any differences in the blood chemistry or not. I think what we are looking at is difference in exposure and the opportunities for exposure. Also, possibly, the role of diet where exposure might be equal; but I am not sure it is clear other than to go beyond that.

Mr. MOLINARI. We heard a great deal about the incidence of sickle cell anemia among the black population. Anemia is one of the symptoms of exposure to lead. Is there any causal relationship there?

Mr. **Dyer**. I don't think it is clearcut. I am not aware of any basis.

Mr. **Molinari**. After hearing the testimony of Mrs. Spears this morning, it seems as though there was a pronounced difference in the incidence of problems after 1981. Yet the smelters, according to the information we have, are 40 years old or thereabouts. Do we have any information indicating that perhaps the equipment was breaking down or that there was some other reason as to why there was an increase in problems, if indeed there was an increase, in the incidence of lead pollution from the smelters?

Mr. **Dyer**. I think the records will show that there have been problems for a long time. Initially, I think the suits were addressed at the area emissions. The city of Dallas did take some enforcement actions against the companies years ago regarding air emissions. I think the problem is that in those cases, soil was not addressed as an exposure mechanism.

Mrs. **Phillips**. But as to a specific breakdown in 1981, I am not aware of any specific breakdown. In fact, the companies had put on air emissions equipment in the seventies; and part of the problem we were faced with here was that the lead in the soil we believed at that time was caused by historical air emissions, so the problem we faced was something that the companies had not created today but had historically done and were doing in compliance with all State, local, and Federal requirements.

That was one of the things we were having to deal with as we looked at our legal theories.

Mr. **Molinari**. You don't believe it was due to any mechanical breakdown or anything that was being done in the operation of the smelters that might have increased the amount of lead emissions?

Mr. **Davis**. To the best of our knowledge, no. Based on the city inspections and State inspections at the facility, both facilities appeared to be in compliance.

Mr. **Molinari**. I have a copy of testimony by Dr. George E. Hardy, Jr., who is Assistant Director in Washington for the Center for Disease Control. This was testimony given on December 2, 1982. He refers to a study in 1973 where 11.1 percent of the children screened in high risk project grant areas were identified with lead toxicity. Then he goes on to say that has dropped substantially.

That study showed 11.1 percent in high risk areas but in Dallas the report indicates that roughly 5 percent of the children showed a lead toxicity problem? I have difficulty in my own mind trying to understand if this was half the incidence in 1973, why is it such a critical problem?

Mr. **Dyer**. Was that 11.7 percent lead toxicity or lead elevation?

Mr. **Molinari**. Lead toxicity.

Ms. **Phillips**. I think the difference is our reference area in Dallas had a—showed zero lead toxicity, and around the smelters we were showing a 5.6. What you are referring to is a national average study. I think that might be the distinction that you are looking for.

Mr. **McAnalley**. I would like to address that. I think that is something we really need to look at. See, what we are trying to do, as I understand it, or what EPA is supposed to do is reduce environmental harm or injury to humans, potential injury.

Now, around Dallas, like in Highland Park, certain areas, down home kids are above 20? Zero.

Dallas doesn't have a lead problem because nearly everything is new. We don't have old painted houses. So, is it OK to say, hey, you know, you only have—your level is lower and therefore it is OK when it could be zero and we know the source?

We are looking for the source—when we do that, we take that information and take it elsewhere and maybe reduce the others, but we are covering it up right here. We have never looked at the wedge.

Besides, that is a 2-mile circle that this average comes from. In other words, on our soil—isn't this right, Norm? We have a little wedge where people live and they sample all the way around.

We thought these people are probably normal. That is all we wanted to know is, if 12 percent in that circle is too high, maybe 100 percent in the wedge is too high.

See, if the—this is like a piece of pie. They are taking the whole pie. That, to me, is criminal; when the experts say look in the wedge, why don't we look in the wedge?

Why don't we keep drawing bigger circles and saying the average in that circle is close to the national average?

Now, none of us understand that in science. If it is still too high, you take a bigger circle. Get the kids over here that have indoor swimming pools that never go outside. That is how you get it down lower, and to go back now that they have removed a bunch of soil—and I heard earlier a mother saying exactly what CDC said when they were afraid and finally you tell them they are OK, they get poisoned again.

Now, that has been shown time and time again in lead poisoning and we don't need to show it again.

Mr. **Molinari**. Did RSR engage in some cleanup work?

Ms. **Phillips**. Yes, sir.

Mr. **Molinari**. Do we know how extensive that clean-up work was?

Ms. **Phillips**. We have a piece of correspondence from them that I would be glad to submit for the record dated September 8, 1981, describing the extent of their cleanup work at the playgrounds and day care centers in the area.

I believe there was one day care center that they were not able to clean up because the day care center would not allow them on their property.

Mr. **Molinari**. Mr. Chairman, I would like to ask that that be entered into the record.

Mr. **Levitas**. Without objection.

[The information received follows:]

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September 8, 1981

Ms. Frances E. Phillips
Acting Regional Administrator
Dallas Regional Office
Environmental Protection Agency
28th Floor
First International Building
Dallas, Texas 75270

Re: Soil Removal by RSR Corporation

Dear Ms. Phillips:

Following the release of EPA's Dallas soil-lead study earlier this year, RSR Corporation advised EPA that it would remove soil located in high risk areas in elementary schools, playgrounds, and day care centers near RSR's plant that had been identified in the study as containing lead in excess of 1000 parts per million (ppm). The sites so identified were Jose Navarro Elementary School, Tipton Park, and Tabernacle Day Care Center. RSR promptly began efforts to contact appropriate officials at the Dallas Independent School District, the Parks and Recreation Department of the City of Dallas, and Tabernacle Day Care Center in order to arrange for further soil sampling and for soil removal and replacement if necessary. The purpose of this letter is to bring you up to date on the progress of RSR's efforts.

RSR retained Mr. V. K. Argento, who had participated in preparing the EPA study, to do additional soil sampling at Jose Navarro School and at Tipton Park while arrangements were being made with authorities with respect to soil removal procedures. Because Tabernacle Day Care Center is on private property, RSR determined not to do further sampling there until approval could be obtained from the day care center's management.

Meetings were held in July with Mr. Paul Barger, Maintenance Administrator for the Dallas Independent School District, and Mr. Philip Huey, Assistant Director of Maintenance and Beautification for the Parks and Recreation Department. Agreement was reached in each instance as to the procedure for soil removal and replacement if that should be shown necessary by the resampling then in progress. Repeated efforts to reach someone in a position of authority at Tabernacle Day Care Center, beginning in June and continuing through August, have been unsuccessful.

By letter dated July 31, 1981, Mr. Argento reported to RSR the findings of his follow-up soil sampling. Of the four samples taken at Jose Navarro School, the one under the swing set contained 1182 ppm lead; the others contained lead at levels of 167 ppm, 69 ppm, and 288 ppm. None of the five samples taken at Tipton Park contained lead approaching the 1000 ppm level; the lead levels in the Tipton Park samples were 164 ppm, 78 ppm, 105 ppm, 229 ppm, and 2 ppm.

Between August 15 and August 18, 1981, RSR's contractor removed and replaced top soil in the area around the swing set at Jose Navarro School. The new soil, which was tested and found not to contain significant quantities of lead or other contaminants, was packed to a depth and degree of firmness at least as great as the removed soil. Mr. Ray Lovejoy of the Dallas Independent School District was present during both the removal and replacement phases of the operation, and expressed his approval of the work performed. Because all of the samples taken from Tipton Park were low in lead content, it was decided that soil removal was not appropriate at that location.

We are disappointed by our inability to obtain cooperation from authorities at Tabernacle Day Care Center. Between June 29 and July 24 at least a dozen phone calls to the Center were made by RSR personnel and myself. Most frequently we spoke with a Mrs. Reagans, who told us that Dr. Reagans was the person with whom we should talk. Repeatedly, we were told that Dr. Reagans was either unavailable or away from the building temporarily, but that a message would be left asking him to return our call. Our calls were never returned. On July 24, Mrs. Reagans advised me that I should state my message in a letter to Dr. R. D. Gray, Chairman of the Board of Tabernacle Day Care Center, and send that letter to Dr. Gray at the Center. I have now sent two letters to Dr. Gray expressing RSR's interest in replacing soil at Tabernacle Day Care Center if necessary; copies of both letters were sent to Dr. Norman Dyer at EPA. We have received no response to either of the letters. RSR has determined that further efforts on its part to contact authorities at Tabernacle Day Care Center would not be worthwhile.

If you would like to discuss RSR's soil removal activities generally or the situation at the day care center in particular, please do not hesitate to call us.

Sincerely,

Robert M Cohan

Robert M. Cohan

RMC:mc

Mr. Molinari. Ms. Phillips, could you just briefly summarize that report for us?

Ms. Phillips. Yes, sir. The report is an advisory—it is a letter from RSR to EPA stating that they are advising us that they removed soil located in high risk areas in elementary schools, playgrounds, day care centers near RSR's plant that had been identified as containing lead in excess of 1,000 parts per million, and it goes on to explain how they sampled, then removed the soil, and placed top soil in the area.

Mr. Molinari. To the best of your knowledge, did they do what they said they were going to do in that letter?

Ms. Phillips. Yes, sir.

Mr. Molinari. The only location where they weren't able to conclude their work was one where they refused to grant the company access?

Mrs. Phillips. Yes, sir.

Mr. Molinari. It would seem then that whether enforcement action was taken or not, at least one of the responsible parties went about doing the cleanup work that region 6 would have recommended.

Ms. Phillips. Yes, sir.

Mr. Molinari. That leads us to the question of whether there was a greater health impact on the children in the area because of poor judgment on the part of Dr. Hernandez.

Dr. Dyer. The cleanup was very limited actually. One elementary school playground, a park, and the boys club park area. There was nothing done about the residential play areas, and so there's a large project area right off from the facility.

There was nothing done about that project area, so you still had highly contaminated soil in most areas with opportunity for exposure.

Mr. Molinari. EPA is generally called in by a city or by a State and CDC is willing to render technical assistance when requested.

I do know from reading the data here today there was some question about the blood sampling that was done by the city of Dallas. I think if I recall correctly the testimony was and the documents indicate that those tests were inconclusive.

Was that because of some problems with the methodology that they used?

Dr. Dyer.

Mr. Dyer. No. It was not due to methodology. It was primarily because it was a self-selected population and citing statistical analysis for random selection, the statisticians concluded that it possibly had sample bias because of self-selection, but they say there was no indication, in looking at the data, of sample bias. It seemed to represent the population there, but from a statistical standpoint, they wanted to do it statistically.

Mr. Molinari. Did there come a point when EPA told the city of Dallas to conduct new testing and did EPA lay out the guidelines they suggested for retesting? Mr. Davis.

Mr. Davis. A couple of points, the first being, there is a popular misconception that the city of Dallas' study was inadequate and we criticized it and couldn't draw conclusions from it.

The reality of the situation is that the city of Dallas study was never intended to be a study. When we were working in early June with the companies, that was going to be the study that was going to provide us answers.

The city at the same time said if the data was inconclusive from the earlier work, they would open their clinics; hopefully we would get voluntary participation and be able to see if there was a problem around the area.

They opened the clinics around June 29 to about July 13 and had a very terrible turnout. I think only five people came in. We were negotiating with the company to do these supplemental surveys. There was a lot of media attention. There was a lot of city council attention.

At that time, the city council made a decision that they needed to open their clinics for concerned citizens to come in to see if there was a blood problem. It was never designed as a study. It was a city concern. They originally planned to test, I believe, 2,000 children within the 2-mile circle—diameter around the smelter. They eventually, I think, ended up testing over 12,000 individuals, not only preschool children which were of concern, zero to 6 years old, but 6 to 17, and also adults.

They also tested adults, people who came from outside the 2-mile area.

So, during this period of time, we were trying to use all the data that was available to us to reach a conclusion. So it was not a study. The report that CDC reviewed EPA put out in April 1982 on the voluntary city program was an evaluation of that study to see if conclusions could be drawn.

A couple of points that I would like to make that have been explained to the regional office and I cannot defend explicitly—experts would have to defend it, but the fallacy in the argument that I have heard about testing of the wedge and the reason for the CDC/EPA study to define the relationship is simply if you have a 1-mile circle—and let's say 2 percent of the individuals in the circle have elevated levels of lead in their blood—generally I am going to guess—if you have the circle, you will increase the concentration by a factor of 4. So you go from a 1-mile radius circle to a half-mile circle, which we did, you get 6 percent of the people with elevated blood lead. You then go into a quarter mile and you increase by a factor of 4 again. So you have 24 percent of the people.

Now, let's take it to a 90 degree wedge. Assume downwind, maximum soil concentration, 90 degrees is one-quarter of 360. Let's multiply it again. Four times 24, whatever. Now, you have 70 percent of the people with elevated levels of blood. OK?

Let's now take the housing development just north of the boys club which is downwind. You get 90 percent. You pick the specific apartment with the maximum concentration; you get 100 percent.

Superficially that is a very good argument that lead in soil is the cause of the problem. You have a 100-percent correlation.

As explained to me, the fallacy in that argument and the reason that the study had to be statistically designed with a confidence level—and I believe the confidence level for this survey was the 95 percent—was that, yes, superficially high soil, high blood; but how do you explain two blocks away where you have the exact same high soil concentration the kids have zero lead toxicity?

So the reason for the scientifically designed study as explained to me is to establish the scientific correlation between blood and soil so that you not only answer the question yes, where you have high blood you have high soil, but where you have high soil do you have high blood?

No, unfortunately, I do not have a copy of the report with me. I gave my only copy to the committee staff, but the technical support document, the technical work that was done by our contractor, RTI, in support of the work analysis by CDC and the peer group review analyzed the correlation between blood and soil.

It is what is known as a correlation coefficient that I think a mathematician would have to explain to you.

Essentially with a correlation coefficient of zero, you have no correlation. You have lead in the soil and you can get anything out. If you have a correlation coefficient of 1, that means you can predict with mathematical certainty this soil level results in this blood level.

The correlation that RTI found when they analyzed soil versus the blood lead in children was a correlation coefficient for both sites, both sites, both RSR and Dixie, the same—and the correlation coefficient was 0.4.

As explained to me, what the 0.4 correlation coefficient means is there is an association. It is not very strong, but there is an association.

So when we analyzed the data, when the task force analyzes the data, they have the Dixie results which show no lead toxicity, but they do show elevated levels of blood lead in children of 1.85; compared to the reference area, an increase.

You go over to RSR. Generally, the same soil levels, a little higher levels, a little further out. You see a 7.2-percent—7.2-increase over 14.7 which is about 50 percent.

So there is a dramatic increase. The soil level at both sites are similar. The difference, the air.

Now, I am not saying that the report is saying that there is no contribution of soil, and I think that the CDC and the peer group panel were very concerned because their recommendation in the final report, even though Dixie showed no lead toxicity, and only showed 1.85 micrograms per deciliter, their recommendation was for both smelter sites because of their concern.

What was their recommendation? Their recommendation, in substance, aside from the regular blood screening and to bring the emissions under control, was to create a barrier between children's exposure to soil, to the environmental sources of lead.

Their recommendation, to look at the high use areas first; day care centers, schools, playgrounds, public housing areas. OK?

That was CDC's recommendation. That is the recommendation that we are working on at the present time with the Texas Air Control Board to bring the source into compliance with the air emission standards and with the city of Dallas who has ongoing negotiations with both companies to clean up within a half mile of both smelters. Because of this recommendation—and it might be that—and I didn't particularly remember this—that CDC may not have addressed their original charter of the direct correlation between soil and blood lead and didn't come up with a number 750, 500, 250, whatever the number is.

There wasn't lead toxicity. They saw an increased level of blood lead that they were concerned about, even though it was small around the Dixie facility and their recommendation was the same.

Reduce it to the maximum amount possible, not to an isopleth level, a line of constant concentration, but to a half mile, because that is where the break point was in the data.

Outside half a mile, not just downwind, but to a half a mile, address the areas the children are playing in. They said that for the first time with scientific certainty, and that is what is happening now.

Why they couldn't draw the same conclusion from the city of Dallas study, I can superficially answer is that it was a voluntary program and they just had blood lead. They didn't have the corresponding soil for the child.

The study was subsequently done and we had 25 people in the field this summer, ten teams: they drew blood lead, they filled out questionnaires on lifestyle to establish the exposure pathway; they sampled front yards for samples, side, back yards. We overlayed a grid on top of the whole area so we could plot isopleths, so we could come up with the answer.

The answer was there was an association, a correlation coefficient of 0.4. But the CDC recommendation, because of their concern, still was to go in and clean up. I think that was a responsible position; a very responsible recommendation based on the information available.

But to second-guess the health professionals and say they should have known that 2 years ago, I am not sure that is fair.

Mr. MOLINARI. I want to thank you, Mr. Davis, for putting this in perspective. Frankly, I think I understand it a great deal better in view of the explanation you have just given.

There are traffic studies indicating that there is some high traffic density in the area. From the research I did preliminary to this meeting, I found that about 98 percent of the airborne lead can be traced to the source. It comes from the combustion of gasoline.

Are we able to identify from the figures that you just referred to as whether they come from the smelters or the high density traffic in the area?

Mr. DAVIS. The study was very carefully designed to do that. Both the reference areas—unfortunately, we are stuck with the roads of the smelters. One of the roads at the smelters, I think, had about 18,000 cars per day.

There is a major intersection at one smelter. The other roadway had 10,000.

The other smelter roadway had about 14,000 cars per day.

Mr. MOLINARI. 14,000.

Mr. DAVIS. 14,000 cars per day a half mile away. Because it was a half mile away, and if you go to the statistics, you see some real strange results. But they were able by multivariant analysis, to show that around the RSR site, the contribution of lead in children's blood was about—and I am just doing it in my head—17 percent from automobile emissions and 83 percent from the smelter.

It is a ratio of 5.5 to 1. It is in the final CDC report. We were able to determine that because you had high traffic areas and low traffic areas. The problem with the Dixie site is you didn't have high traffic close to the smelter. It was a half mile away.

So we weren't able to identify the contribution of the Dixie smelter from automobiles, but common sense tells you that since the roadway is a half mile away, and you didn't have lead toxicity, but you had an increase in blood lead, though very small, I am going to make the assumption that 90 to 95, 98 percent of the elevated levels of blood lead was due to the smelter, due to the soil.

Mr. **MOLINARI**. Thank you very much. You have enlightened me a great deal. I note that Mr. McAnalley seems anxious to speak.

Mr. **MCANALLEY**. My first degree was in math. I have had doctoral level degrees in statistics. The thing we have to understand is that we really don't understand all about statistics. There is a book written on how to lie with statistics.

I have done statistics that prove my dead rats are alive. No matter how much you show those statistics, they don't get up.

Mr. **MOLINARI**. We are trying to get rid of some of those dead rats.

Mr. **MCANALLEY**. The point is we had a huge circle, and you need to go over and look. If you look at this picture here, we had a huge circle. We had this wedge. We wanted to look at it. Right now—I drove by there the other day.

Most of those buildings have several windows boarded up. You know, they have got either the people behind—behind boarded-up windows and can't see out or there is nobody in there.

If you go back and do this great elaborate study after everybody has gone and show no problem around that area, that is a disgrace to science for anyone to even be involved in something like that.

Some of my medical—some of my peers think—they mentioned that the other day. That is a disgrace; for it to even be presented is a disgrace.

It must be a coverup. That is the only reason I can think that you would present a study like that.

I would like to add here is the smelter. Across the street you can throw a rock and hit it from the boys club. Now the boys club had sod all around it. They planted grass and there are sprinkler systems around there.

What is nice now is the kids who want to come to that boys club are going to have to walk by the poison, walk through the poison, play, and walk home.

You go over here and sample them. They are high because they walked through the poison to play. That is stupid.

To present a study like that is stupid, Fran. You are part of the coverup.

Mr. **MOLINARI**. Well, on that note, Mr. McAnalley, I thank you for your candor.

Apparently there are serious differences of opinion here.

I have no further questions, Mr. Chairman. I will turn it back to you.

Mr. **LEVITAS**. Thank you, Mr. Molinari. Mr. Rowland.

Mr. **ROWLAND**. Thank you, Mr. Chairman.

It seems I have heard two different things here. I would like to get clarification on it.

Dr. Davis says the correlation between the soil and the blood according to the correlation coefficient was low or soft. Dr. Dyer had said earlier that the study did not answer the basic questions, did not make the correlation.

I want to get some clarification on that if I can.

Mr. DYER. I think the correlation Dr. Davis is speaking of was some preliminary correlations that are not included in the final report, at least the final report issued February 1, 1983, does not include those soil lead/blood lead correlations.

Mr. ROWLAND. Is that what you said?

Mr. DAVIS. What Dr. Dyer indicated is true. The report that I was referring to was the RTI report that the basic information provided to CDC and the medical panel—and they selected the appropriate tables and information they wanted to use.

My understanding of why that table was not in the final report was that they considered the blood lead data, and this recommendation took the soft correlation into consideration on their recommendation for addressing the soil out to half a mile, but I think you would have to talk to the author of the report.

Mr. ROWLAND. Thank you, Mr. Chairman.

Mr. LEVITAS. Thank you, Mr. Rowland.

Ms. Phillips, is it correct that the Dallas lead sites were not designated as Superfund sites?

Ms. PHILLIPS. That is correct.

Mr. LEVITAS. Was any consideration given to designating them as Superfund sites?

Mr. DAVIS. Whenever we were dealing with Dallas lead, the enforcement options including Superfund was always a potential. I do not have the date of the correspondence, but one of the ladies in the Dallas area affected by the smelter wrote the Texas Department of Water Resources. You have to realize their response was really taken out of context.

They indicated that Superfund would not appear to apply to a facility such as this, but that they had not checked with the regional office, and I would imagine that was a cursory response.

The region always considered the possibility of using Superfund. Obviously, the State didn't nominate the site, but we did consider this as a potential Superfund site, went through the ranking process for it, and had the number in our back pocket if either the State or ourselves decided to go forward with a Superfund action.

Mr. LEVITAS. How did it rank?

Mr. DAVIS. I don't know the actual cutoff score for Superfund, but it was very close or slightly above.

Mr. LEVITAS. I have an option paper of April 8, 1982, from Dr. Bracken to Dr. Hernandez. Under the heading, "Remedial Action," there is a rather extensive discussion that EPA could consider enforcement action under various imminent hazard provisions of the statutes they administer including RCRA.

This could result in cleanup by the smelter owners. Another option would be to extend Superfund money to cleanup the site.

This would require placing the site on the national priority list, et cetera.

Do you know why that decision was not made—why a decision was made not to do that? Does anybody at the table—did anybody——

Ms. Phillips. I know initially—and I am talking about in April and May 1981, initially we did not pursue Superfund because the program was very new at that time. We didn't have our first list, and we were concerned that we thought Superfund was to be used for abandoned sites and we didn't have a classical Superfund case.

So that was our initial reasoning.

Mr. Levitas. This is April 1981.

Ms. Phillips. Right.

Mr. Davis. That is the point. Once the decision is made on June 1, 1981, not to proceed with a cleanup or an action level and the need for additional studies, we are now on the course of proceeding with additional studies. We had been negotiating with the companies for studies. We broke off negotiations because the city of Dallas volunteer programs were underway. CDC analyzed the report and came up with the need for additional studies.

We then do the additional study. It is now February 1, 1983. The recommendation comes out. We are proceeding with the city and State to negotiate with the companies. Don't forget, under Superfund we have to send a responsible party letter out before we dip into the fund and give them the opportunity to do what we are requesting them to do.

Until then, there is no need for formal enforcement action.

Mr. Levitas. Let me see if I understand all of what Ms. Phillips said and what you just said. That goes back—what you just said goes back to the decision of June 1, 1981. In retrospect, do you agree with that decision?

Mr. Davis. I, no——

Mr. Levitas. Let me rephrase the question. It sounds to me from what you are saying that if you had to call the shot in that period of time, even in retrospect you would make the same decision.

Mr. Davis. I would stand behind the regional decision, the recommendation in May 1981. I thought it was right then. I think it is right now. And I understand where the deputy administrator was coming from, at least from a scientific standpoint. I can understand it. I didn't say I agree with it.

Mr. Levitas. It was beginning to sound as if you agreed with that philosophy. I think it ought to be very clear from your point of view for the record that you don't agree with that decision. Since they can't get the nod of your head, you ought to say yes or no.

Mr. Davis. Yes.

Mr. Levitas. Thank you.,

Mr. McAnalley. If I can add on that, based on that particular model if you had an emission site, say a pipe running along here, and a family was being killed. Statistically, it would show no problem, see. So what that tells us is our statistics are not adequate. We just simply don't know everything. That is what scientists need to understand. I was asking one question when I got my first master's degree. I didn't know. They said, congratulations, Dr. McAnalley, you passed. Because nobody else knew, either. But they want a scientist to say he knows what he doesn't know.

That is what we have here. Statistically those studies have been done by CDC, ASARRO. They have been done by Silver Valley, and we know the relationship between blood and soil. That is known.

What we wanted to do here, if I was one of the family members or they were my children, I wouldn't want them to statistically prove it. What we want to do is go in and help the kids, no matter where it came from, because I heard women describing those symptoms before and those are the symptoms of lead poisoning. I also heard of a dog, or several dogs, people around there couldn't keep a dog. You know this veterinarian said they all died from kidney poisoning. Well, that is lead poisoning.

So based upon that we knew there was a problem there. Until we knew how to statistically do it, we ought to be able to go in and fix the problem.

Mr. LEVITAS. Yes, Mr. Whittington?

Mr. WHITTINGTON. Mr. Chairman, I would like to respond to the allegations that Dr. McAnalley made with respect to coverup. It is apparent that he has rather strong opinions. Other people have strong opinions. In point of fact, I received an unsoliciated call in my office, Tuesday, I believe, from the former city health officer of the city of Dallas, who said, and I quote him I think accurately, a Dr. Barry, whom I have never met, that the only coverup in this instance is people trying to convert a nonproblem into a problem. So you do have people with strong opinions on both sides.

I would like to say that with respect to the presentation of the data which Dr. McAnalley characterized as shameful or some such, that the data was carefully presented both in the context of what was considered to be a National average, which I think is a valid presentation in the sense that it places the matter into context. It was also carefully placed into perspective with respect to the reference area located in the city of Dallas for that specific reason. The recommendations made by the CDC address both the air and the soil, and they base those recommendation on the basis of the increase in blood lead over the reference area. So the data was in my judgment, anyway, fairly presented.

I would say it makes little difference to the percentage of the kids this way or that way because the recommendations are for remedial measures to be taken, and those measures are underway, or shortly will be. So we are taking a while to get there but we are going to get there.

Mr. LEVITAS. I appreciate your making that statement, that it took a while to get there. I want to get back to that.

Mr. WHITTINGTON. Also, if I could, Mr. Chairman, I would like to address one other thing. The concern that you have that somehow or other health studies would be part of our standard operating procedures in connection with determining whether to clean up, sue, or so forth, and certainly in region VI, this is the only case where this has been done. And we have taken other enforcement actions, cleanup actions and the like.

Mr. LEVITAS. We very likely will be exploring that further in another context.

Before we conclude the hearing I said I would announce that we will be receiving the enforcement sensitive documents on all the first six sites we requested. We will receive them this afternoon at approximately 3 o'clock, including Bridgeport, NJ, Seymour, IN, PAS, Oswego, NY, Ottati; and Goss in New Hampshire, Keefe Environmental Services, New Hampshire, and Stringfellow, CA.

Pursuant to the agreement that I have now reached with the administration, our staff will promptly proceed to examine the files in place on the other sites and these sites as well that we are going to be focusing on. I just want to conclude these hearings today, and there are still elements we need to put together. We are going to hear from—we are going to get some information for the record from representatives of EPA headquarters who have been involved in this.

But to me, what happened in this particular matter is one of the most unforgiveable examples of how the public health was not protected, that you don't let people, particularly children, remain at risk where you have the potential of a health hazard. If you are going to err, you err on the side of protecting the public. And nothing would have been better or happier for everybody concerned as if the action was taken in June 1981 and you did your further studies and you found out that it was unnecessarily taken. That would have been an awful lot better than leaving children at risk for 18 months. And the other day in discussing this with the acting administrator, I made the same point.

That policy is a policy which does a total discredit to the entire mission of the Environmental Protection Agency. I hope that, among other things, Mr. Ruckelshaus, the incoming Administrator, will never again let that type of policy be the policy of the Environmental Protection Agency. I feel very strongly about that. And I think that it is a disgrace to have approached the problem where, with the evidence available, with the information available, and with the willingness on the part of the contaminators to begin to do something, and the decision made by the regional officials who had been working with this to get on with that cleanup, that that was stopped dead in its tracks, and 18 months later, or as you said, Mr. Whittington, after a while, we came to that conclusion. I would not have wanted my child to be exposed to those levels of lead for 18 months while people were trying to get the scientifically perfect answer of what to do, because I fear, as Mr. Molinari pointed out in his questions, that that decision lead to the exposure of children that could have been avoided had prompt action been taken when the decision was made in June 1981.

The record will be kept open for 2 weeks for additional comments from any of the witnesses today and others to whom questions may be submitted.

There being no further business to come before the subcommittee, the subcommittee stands adjourned subject to the call of the Chair.

[Whereupon, at 2:20 p.m., the subcommittee was adjourned, to reconvene subject to the call of the Chair.]

[The following was received for the record:]

TO: Bill Hathaway, Enforcement Division
FROM: Bill McAnalley, Regional Expert in Toxicology
SUBJECT: Lead Poisoning
DATE: September 4, 1981

Symptoms and signs of chronic lead poisoning can be divided into six categories: gastrointestinal, neuromuscular, central nervous system, hematological, renal and others. They may occur separately or in combination.

Clinical diagnosis of lead poisoning is easily missed in the absence of known abnormal exposure. The concentration of lead in blood is the best indication of recent absorption of the metal.

As mentioned above in the first paragraph, lead causes abnormalities in the formation of the blood cells. Specifically, lead blocks the incorporation of iron into the protoporphyrin ring to make heme, which would then form hemoglobin, and this can cause a build-up of protoporphyrin in the blood. Also, iron dificient diets can increase blood protoporphyrin. Consequently, elevated blood protoporphyrin is a symptom of malnutrition, the most frequent childhood disease in the U.S., and lead poisoning, the second most frequent childhood disease in America. Elevated protoporphyrin has been used extensively by C.D.C. to screen for these two childhood diseases.

An elevated protoporphyrin indicates malnutrition, lead poisoning or a combination. The blood lead concentration must be determined to distinguish malnutrition from lead poisoning.

In the attached figure, the blood lead concentration is plotted on the x-axis and the zinc protoporphyrin is plotted on the y-axis. If a line is drawn at 50 µg/100 ml blood zinc protoporphyrin to signal possible lead poisoning, twelve out of twelve of the children with blood lead concentrations above 60 µg/100 ml are identified. This indicates that the zinc protoporphyrin test could be used to identify 100% of the children with blood lead concentrations above 60 µg/100 ml. The percentage of children with zinc protoporphyrin test above 50 in each lead group are listed below:

Group	Lead Blood Concentration µg/100 ml blood	Percentage of Children Identified by Elevated Zinc Protoporphyrin Test
I	0 - 30	7.4% (4/54)
II	30 - 40	18.0% (9/50)
III	40 - 50	50.0% (23/46)
IV	50 - 60	84.0% (21/25)
V	Above 60	100.0% (12/12)

The preliminary lead study by Dr. Barry of the Dallas Health Department, found 100% of the screened children in the 30 to 40 range (Group II). If half the children around the lead smelter are in the 30 to 40 range and all the other children are below 20, only 9% of the children would be identified by the elevated zinc protoporphyrin test as having elevated lead. When compared to the national average it would appear that the children living around the smelter had a normal lead exposure when in fact 50% are above the acceptable limit of 30 µg/100 ml blood.

It is my recommendation that blood lead determinations should be used to evaluate lead absorption.

with its acceptance by the Public Health Service as an alternative to blood lead testing in screening children for lead poisoning. Protoporphyrins accumulate when the insertion of iron into the protophyrin ring to form heme is blocked by lead. Their build-up in blood is roughly proportional to the blood lead level; but more important, their measurement can serve as a monitor of the toxic effects of lead absorption.

It has recently been discovered,[23] or perhaps rediscovered, that the protoporphyrin in FEP is not really free but is mostly bound with zinc to form zinc protoporphyrin (ZP). The measurement of ZP can be done simply, rapidly, without fear of contamination, directly on a drop of blood, and offers a better and more convenient test for the effects of lead toxicity than FEP. We have demonstrated this persuasively for children; and have also applied the test to adult populations of lead workers, Fig 6.

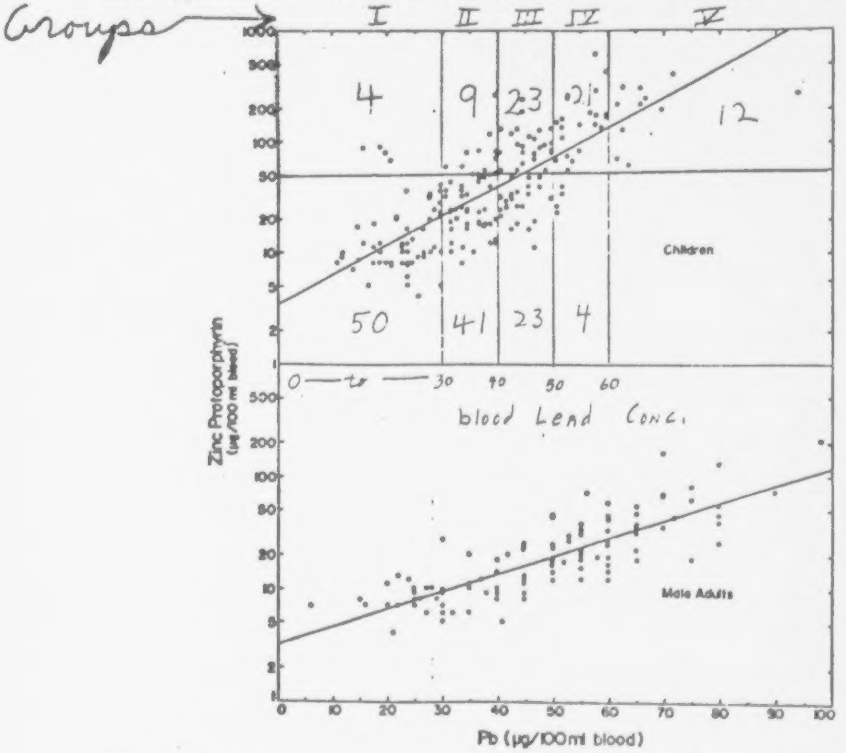

Figure 6 Relationship Between ZP and Blood Pb (23)
Adapted from Lamola, A., Joselow, M., Yamane. T.L., Clin. Chem. 21:92-97, 1973

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION VI
1201 ELM STREET
DALLAS, TEXAS 75270

April 6, 1983

REC'D APR 7 1983
Subcommittee on Investigations
and Oversight
Committee on Public Works
and Transportation

Honorable Elliott H. Levitas, Chairman
Subcommittee on Investigations and Oversight
Public Works and Transportation Committee
House of Representatives
Washington, DC 20515

Dear Mr. Chairman:

During your hearing of March 24, 1983, on Lead Contamination Around Three Smelters in Dallas, Texas, you asked me to submit for the record several documents from our regional files. Accordingly, I am enclosing the following:

1. The letter dated April 21, 1981, from John DePaul, RSR Corporation, to Dr. Norm Dyer, EPA, Region 6.

2. The press release dated June 1, 1981, as proposed to be released through Eddie Lee.

3. The press release dated June 1, 1981, as released through Deborah DeBerry-Kershaw.

4. The press release dated June 1, 1981, as released through John DePaul, RSR Corporation.

5. The letter dated September 8, 1981, from Robert M. Cohan, attorney for RSR Corporation, to Frances E. Phillips, EPA, Region 6, relating to soil removal by RSR.

6. The November 2, 1981, memorandum from Walter Kovalick, Jr. to Al Davis, titled, "Status Report of Lead/Smelters Study Group."

Finally, as is evidenced from the enclosed memorandum to Dr. Allyn Davis, we diligently searched the files and were unable to locate the "speaking points" I used to convey our regional concerns to Dr. Hernandez in early June. I imagine that since they were intended for use as personal notes, they were never included in the official file and were thrown away long ago. However, the essence of these "points" was reiterated in the June 29, 1981, Status Report prepared by the Region for Dr. Hernandez. See, particularly, sections "III. Health Effects and Significance of Environmental Exposures, B. Action Soil Level" and "V. Policy Ramifications, C. Setting a Precedent for Demonstrating Adverse Health Effects", pages 11 and 14.

I hope that I have included everything that you requested. If you need any further information please call me at 214-767-2600 or have your staff contact Ms. Barbara Goetz at 214-767-2605.

Sincerely yours,

Frances E. Phillips

Frances E. Phillips
Deputy Regional Administrator

Enclosures

RSR
CORPORATION

April 21, 1981

Dr. Norman Dyer
Chief, Pesticides & Toxic
Substances Branch
Environmental Protection Agency,
Region 6
First International Building
Dallas, Texas 75270

Dear Dr. Dyer:

RSR Corporation (RSR) understands that the U.S. Environmental Protection Agency (EPA) has contracted for the monitoring of lead and cadmium around several lead emission sources in the Dallas area, including RSR's lead smelting facility. Although RSR has not received a copy of the report and has not had an opportunity to study its contents, we understand that EPA intends to release the report to the public in the near future. We have asked to meet with you today to discuss release of the report because we believe that the public interest will best be served by a fair and accurate presentation and by an expeditious response to any problem that may be found to exist.

The Lead and Cadmium Study

Request for Quotations No. R6-80-020, issued August 25, 1980, indicated that EPA was seeking data concerning lead and cadmium levels near Dallas area lead emission sources. This data was to be obtained by comparing soil, house dust and tap water samples collected in neighborhoods within a two mile radius of the selected emission sources with control samples collected from neighborhoods not affected by a major source of lead.

RSR understands that EPA has received a report from the contractor containing the data requested by EPA. Although we have not been able to obtain a copy of the report, we understand that it indicates that soil samples from neighborhoods near the emission sources, including those from neighborhoods near RSR's smelter, contained higher levels of lead than the control samples.

RSR Corporation

RSR is committed to helping maintain a clean and healthy environment. From 1975 through 1980, RSR spent almost six million dollars in Dallas alone for pollution control. RSR has installed the most advanced available technological improvements to minimize lead emissions from its smelting facilities.

Corporate Offices 1111 West Mockingbird Lane Dallas, Texas 75247
Telephone (214) 631-6070 Telex 732-402

RSR is in compliance with all federal, state and local emission standards and requirements. All information available to RSR up to this time indicates that emissions from its Dallas smelter pose no threat to health or the environment. We understand that the City of Dallas has been testing blood lead levels of children in the city on an on-going basis; the City has not given any indication that the tests show high lead levels in children living near RSR's plant. Nevertheless, if soil in neighborhoods near RSR's plant contain high concentrations of lead and those concentrations pose a threat to health or the environment, then RSR wants to take all reasonable steps to alleviate the problem.

To the extent high concentrations of lead may exist in soil near RSR's plant, RSR believes those concentrations result not from current emissions, but from accumulations over some 45 years and from more substantial emissions in years prior to the advent of stringent emission standards and prior to the time the plant was owned by RSR. Those facts do not diminish RSR's desire to alleviate any problem that may exist.

The Public Interest Will Best Be Served By A Balanced Presentation Of The Report

RSR believes that meaningful remedial action can be taken only if the responsible parties have an understanding of the nature and extent of the problem. We further believe that the chances of achieving meaningful remedial action will be enhanced if whatever action is deemed necessary can be taken with the cooperation and understanding of residents of the affected neighborhoods.

RSR urges EPA to share the contractor's report with RSR at this time, prior to public release, to enable EPA and RSR to develop a better understanding of the nature and extent of the problem and to minimize the chances of inaccurate or unbalanced reporting. Because we believe that the understanding and support of affected residents can best be oftained if undue alarm is not created. RSR urges EPA to exercise great care in the manner in which it releases to the public the results of the study. Specifically, RSR requests an opportunity — prior to public release of the results — to review the contractor's report with EPA and to share with EPA its information as to the nature and extent of the threat, if any, to health and the environment.

Possible Approaches To The Problem

Without further knowledge of the findings of the EPA study, RSR is not in a position to know exactly what steps might be necessary to deal with the problems identified therein. RSR, however, is prepared to work with EPA, the contractor, and other technical experts to develop reasonable approaches and solutions.

In some circumstances, it may be necessary to remove the topsoil and replace it with non-lead-bearing soil. In other situations, it may be necessary to turn over the top layers of soil and mix them with layers underneath. In still other circumstances, it may be adequate to cover the existing soil. The levels of lead concentration, the depths at which the lead has been found, and the use of the land will all have a bearing on the proper approach. Obviously, it will be necessary to review and evaluate the report before specific remedies can be confirmed.

In sum, RSR requests the opportunity to work together with EPA to achieve a meaningful solution to any problem that may have been identified by the study. In that regard, RSR urges that EPA carefully consider the manner and timing of its release of the study in order to avoid unnecessary alarm in the community and unnecessary polarization of the parties at the expense of cooperative efforts to protect health and the environment.

Thank you for your cooperation and for providing us this opportunity to meet with you.

Very truly yours,

John A. DePaul

John A. DePaul
Vice President
Technical Services/Engineering

JAD/br

United States
Environmental Protection
Agency

Region 6
Office of Public Awareness (6AP)
First International Building
1201 Elm Street
Dallas, TX 75270

Arkansas
Louisiana
New Mexico
Oklahoma
Texas

EPA

Environmental News

EDDIE LEE
(214) 767-2630

FOR IMMEDIATE RELEASE JUNE 1, 1981

The U. S. Environmental Protection Agency (EPA) today released an EPA funded study, investigating lead and cadmium levels near industrial sources in Dallas. The study shows elevated lead levels in soil near three lead and battery plants.

Samples of lead and cadmium were taken from residential neighborhoods and schools within two miles of Dixie Metals Company, NL Industries, RSR Corporation and General Battery Company and within a reference area remote from known sources of lead and cadmium. Analysis of indoor dust and tap water samples revealed no significant problems with respect to lead or cadmium. Analysis of soil samples indicated that, although cadmium levels were not significantly high, lead levels did show marked elevation in the test areas.

The study shows soil lead levels in the test areas ranging from an average of 131 parts per million (ppm) to 1,253 ppm. The average soil lead level in the reference area was 54 ppm. A 1978 study of lead levels in soil near major traffic arteries in allas indicated an average of 128 ppm.

EPA has begun meeting with State and local officials and representatives of the companies to determine the existence and the scope of any environmental problem and to discuss possible corrective actions. EPA has no evidence of medical injury to residents of the test area.

(more)

Dallas city health officials are reviewing historical blood lead information collected during the past few years through the Early Periodic Screening, Diagnosis and Treatment Program and the Children and Youth Clinic in West Dallas to determine if there is any correlation between the elevated soil lead levels and any increased blood lead levels of children residing in the study areas. No evidence, to date, of any lead concentration justifying medical intervention has been found. However, a number of children, not necessarily living in the areas presently being studied, have exhibited lead concentrations above what is considered normal levels.

Frances E. Phillips, Acting Regional Administrator of EPA for the five state area including Texas, says, "We anticipate the continued cooperation of the companies, and commend RSR Corporation for taking the lead in the voluntary commitments made by the companies involved."

In response to the study, RSR will develop, with EPA, an expanded soil sampling program to determine the extent and magnitude of the environmental problem around its facilities, if any; and support additional biological monitoring to the extent deemed necessary. In addition, RSR will take remedial measures to reduce the soil lead levels in high risk areas such as playgrounds, day care centers, and school yards, where lead concentrations are over 1,000 ppm.

Dixie Metals Company has indicated a willingness to cooperate in corrective measures where needed, including an expanded soil sampling program and the cleanup of soil areas if necessary. They have also indicated a willingness to financially support needed additional blood sampling.

The boundaries of the two areas of further study are as follows: The first test area, located in south Dallas, within a two mile radius downwind of Dixie Metals and NL Industries facilities is divided into two sections. The first section is located on the north flood plain of the Trinity River bounded by Lamar on the south, I-45 on the east, AT&SF Railroad on the west and by Ervay and Forest on the north. The second section is located on the south flood plain of the Trinity River bounded by the Trinity on the north, Sargent Drive on the east, Cedar Crest Blvd. on the west and on the south by Kiest, Rector and Nolen.

(more)

The second test area, in the vicinity of RSR, located in west Dallas, is bounded by the Trinity River on the north, Norwich on the west, T&P Railroad on the south and Kingsbridge on the east.

To assist those living in the test areas who are concerned about possible lead effects, EPA and Dallas health officials advise them to contact their family doctor or Dr. Lowell Berry, Dallas City Health Department, at (214) 670-6634, if any symptoms appear. According to Dr. Norman Dyer, Chief of EPA's Hazardous Materials Branch, the most common symptoms of lead poisoning are anemia, severe intestinal cramps, paralysis of nerves (particularly in the arms and legs), loss of appetite and fatigue. Immediate remedies are available and individuals can be detoxified with proper medical care.

Persons wanting additional information about the study should contact Dr. Norman E. Dyer, EPA, at (214) 767-2734.

#

United States
Environmental Protection
Agency

Region 6
Office of Public Awareness (6AA)
First International Building
1201 Elm Street
Dallas, TX 75270

Arkansas
Louisiana
New Mexico
Oklahoma
Texas

EPA

Environmental News

DEBORAH DeBERRY-KERSH
(214) 767-2630

FOR IMMEDIATE RELEASE JUNE 1, 1981

The U. S. Environmental Protection Agency (EPA) Region 6 today released a preliminary study, funded by the Agency, investigating lead and cadmium levels near industrial sources in Dallas. The study shows elevated lead levels in soil near three lead and battery plants.

Samples of lead and cadmium were taken from residential neighborhoods that lie in a test area within two miles of Dixie Metals Company, NL Industries Inc., RSR Corporation and General Battery Company and within a reference area remote from known sources of lead and cadmium. Analysis of indoor dust and tap water samples revealed no significant problems with respect to lead or cadmium. Analysis of soil samples indicated that, although cadmium levels were not significantly high, lead levels in two test areas did show elevation above levels in the reference area.

The study shows soil lead levels in the test areas ranging from an average of 131 parts per million (ppm) to 1,253 ppm. The average soil lead level in the remote reference area was 54 ppm.

EPA has begun meeting with State and local officials and representatives of the companies to determine the existence and the scope of any environmental problem and to discuss possible corrective actions. EPA has no evidence of medical injury to residents of the test area.

(more)

Dallas city health officials are reviewing historical blood lead information collected during the past few years through the Early Periodic Screening, Diagnosis and Treatment Program and the Children and Youth Clinic in west Dallas to determine if there is any correlation between the elevated soil lead levels and any increased blood lead levels of children residing in the study areas. No evidence, to date, of any lead concentration justifying medical intervention has been found.

In response to the study, RSR will develop, with EPA, an expanded soil sampling program to determine the extent and magnitude of the environmental problem around its facilities, if any; and support additional biological monitoring to the extent deemed necessary. In addition, RSR has indicated it will take remedial measures as necessary to reduce the soil lead levels in high risk areas around its plant.

Dixie Metals Company has indicated a willingness to cooperate in corrective measures where needed, including an expanded soil sampling program and the cleanup of soil areas if necessary. They have also indicated a willingness to financially support needed additional blood sampling.

Frances E. Phillips, Acting Regional Administrator of EPA for the five state area including Texas, says, "The voluntary commitment and assistance of the companies involved is commendable. RSR Corporation in particular took the lead in this process."

The boundaries of the two areas of further study are as follows: The first test area, located in south Dallas, within a two mile radius downwind of Dixie Metals Company and NL Industries Inc. facilities is divided into two sections. The first section is located on the north flood plain of the Trinity River bounded by Lamar on the south, I-45 on the east, AT&SF Railroad on the west and by Ervay and Forest on the north. The second section is located on the south flood plain of the Trinity River bounded by the Trinity on the north, Sargent Drive on the east, Cedar Crest Blvd. on the west and on the south by Kiest, Rector and Nolen.

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Persons wanting additional information about the study should contact Dr. Norman E. Dyer, EPA, at (214) 767-2734.

#

FOR IMMEDIATE RELEASE

June 1, 1981

Contact: John DePaul...(214)631-6070

DALLAS, TEXAS -- RSR has become aware of the Environmental Protection Agency's recent study of lead and cadmium concentrations near Dallas area battery recycling and production plants and of the blood testing program of the City of Dallas.

RSR is encouraged by the fact that authorities have found no existing health problem, and is particularly pleased by Dallas Health Department studies that have revealed no cases of blood lead toxicity in 13,000 children tested since 1977. Because RSR places the highest value on the health of its employees and its neighbors, the company has made environmental controls a major priority: RSR's Dallas plant is currently in compliance with all applicable EPA and state environmental regulations. In addition to the blood test results, RSR is pleased to learn that the EPA study has found no problem to exist with respect to cadmium levels and no problem with respect to lead levels in tap water near RSR's facility. These findings would be consistent with RSR's sound environmental practices.

RSR understands that the EPA study may show that certain areas around the facilities examined, including RSR's Dallas plant, contain soil with higher than normal levels of lead. Even though no current health problem has been found to exist, RSR recognizes the EPA's concern that high lead levels in soil might pose a hazard in the future if not corrected. Therefore, RSR intends to work closely with EPA and local officials to avert potential dangers. Specifically, RSR will clean, remove or cover contaminated soil in high risk areas near its plant. In addition, RSR will develop with the EPA an expanded soil sampling program to determine the extent and magnitude of the environmental problem around its facilities, if any; and will support additional biological monitoring to the extent it is deemed necessary.

According to a company spokesman, RSR is undertaking these actions as a precautionary measure because of its concern for the well-being of the community. RSR's current recycling operations are not a significant contributor to high lead levels in soil; rather these levels reflect build-up over time. The RSR Dallas facility has occupied the same site for almost 50 years, and has long been in an area of heavy automobile traffic. If there is an accumulation of lead in any soil around the plant it more than likely was deposited many years ago. before the advent of advanced pollution control systems, current environmental regulations, or the wide spread use of unleaded gasoline in automobiles.

RSR believes that the EPA serves a very important function by identifying environmental problem areas, even those caused by earlier practices. RSR looks forward to working with the agency in its effort to safeguard the natural environment and the people who live and work near the company's facilities.

RSR prides itself on its pollution control achievements and considers its plants to be a positive contributor to the betterment of the natural environment through the very nature of its business: recycling scrapped batteries into a useable material, thereby eliminating the need to dispose of between 50- and 70-million scrapped batteries each year.

1600 ONE DALLAS CENTRE
DALLAS, TEXAS 75201
(214) 741-0361

WASHINGTON OFFICE
1700 NINETEENTH STREET, N.W.
WASHINGTON, D.C. 20036
(202) 828-1200

ROBERT M. COHAN
PATRICK R. COWLISHAW
RESIDENT ATTORNEYS

11 A.M.
AIR & HAZARDOUS MATERIALS DIV.

6A
6D

RECEIVED
SEP 9 4 33 PM '81
EPA
REGION VI
OFFICE OF REGIONAL ADMINISTRATOR

September 8, 1981

Ms. Frances E. Phillips
Acting Regional Administrator
Dallas Regional Office
Environmental Protection Agency
28th Floor
First International Building
Dallas, Texas 75270

Re: Soil Removal by RSR Corporation

Dear Ms. Phillips:

Following the release of EPA's Dallas soil-lead study earlier this year, RSR Corporation advised EPA that it would remove soil located in high risk areas in elementary schools, playgrounds, and day care centers near RSR's plant that had been identified in the study as containing lead in excess of 1000 parts per million (ppm). The sites so identified were Jose Navarro Elementary School, Tipton Park, and Tabernacle Day Care Center. RSR promptly began efforts to contact appropriate officials at the Dallas Independent School District, the Parks and Recreation Department of the City of Dallas, and Tabernacle Day Care Center in order to arrange for further soil sampling and for soil removal and replacement if necessary. The purpose of this letter is to bring you up to date on the progress of RSR's efforts.

RSR retained Mr. V. K. Argento, who had participated in preparing the EPA study, to do additional soil sampling at Jose Navarro School and at Tipton Park while arrangements were being made with authorities with respect to soil removal procedures. Because Tabernacle Day Care Center is on private property, RSR determined not to do further sampling there until approval could be obtained from the day care center's management.

Meetings were held in July with Mr. Paul Barger, Maintenance Administrator for the Dallas Independent School District, and Mr. Philip Huey, Assistant Director of Maintenance and Beautification for the Parks and Recreation Department. Agreement was reached in each instance as to the procedure for soil removal and replacement if that should be shown necessary by the resampling then in progress. Repeated efforts to reach someone in a position of authority at Tabernacle Day Care Center, beginning in June and continuing through August, have been unsuccessful.

By letter dated July 31, 1981, Mr. Argento reported to RSR the findings of his follow-up soil sampling. Of the four samples taken at Jose Navarro School, the one under the swing set contained 1182 ppm lead; the others contained lead at levels of 167 ppm, 69 ppm, and 288 ppm. None of the five samples taken at Tipton Park contained lead approaching the 1000 ppm level; the lead levels in the Tipton Park samples were 164 ppm, 78 ppm, 105 ppm, 229 ppm, and 2 ppm.

Between August 15 and August 18, 1981, RSR's contractor removed and replaced top soil in the area around the swing set at Jose Navarro School. The new soil, which was tested and found not to contain significant quantities of lead or other contaminants, was packed to a depth and degree of firmness at least as great as the removed soil. Mr. Ray Lovejoy of the Dallas Independent School District was present during both the removal and replacement phases of the operation, and expressed his approval of the work performed. Because all of the samples taken from Tipton Park were low in lead content, it was decided that soil removal was not appropriate at that location.

We are disappointed by our inability to obtain cooperation from authorities at Tabernacle Day Care Center. Between June 29 and July 24 at least a dozen phone calls to the Center were made by RSR personnel and myself. Most frequently we spoke with a Mrs. Reagans, who told us that Dr. Reagans was the person with whom we should talk. Repeatedly, we were told that Dr. Reagans was either unavailable or away from the building temporarily, but that a message would be left asking him to return our call. Our calls were never returned. On July 24, Mrs. Reagans advised me that I should state my message in a letter to Dr. R. D. Gray, Chairman of the Board of Tabernacle Day Care Center, and send that letter to Dr. Gray at the Center. I have now sent two letters to Dr. Gray expressing RSR's interest in replacing soil at Tabernacle Day Care Center if necessary; copies of both letters were sent to Dr. Norman Dyer at EPA. We have received no response to either of the letters. RSR has determined that further efforts on its part to contact authorities at Tabernacle Day Care Center would not be worthwhile.

If you would like to discuss RSR's soil removal activities generally or the situation at the day care center in particular, please do not hesitate to call us.

Sincerely,

Robert M. Cohan

Robert M. Cohan

RMC:mc

Thomas
West J.W
File

JUL 1 7 1981

Mr. Homer P. Hine
Chief Chemist
RSR Corporation
1111 W. Mockingbird Lane
Dallas, Texas 75247

Dear Mr. Hine:

Your letter of June 26, 1981 requested the inspection report and analytical results from the June 11, 1981 inspection of your Dallas facility by Ecology and Environment, Inc..

The inspection report compiled by Ecology and Environment (E and E) includes the results of any samples collected during the inspection. The analytical results from samples collected at your facility are not yet complete, so we have not received a report from E and E. As soon as the report is received by this office, we will forward a copy to you.

If you have any questions on this matter, please call Mr. Larry Wright at (214) 767-3274.

Sincerely,

ORIGINAL SIGNED BY

Robert B. Elliott, Chief
General Enforcement Branch, 6EG

bcc: Norm Dyer, 6AH-P ✓
TX06971

6EGH:WRIGHT:RN:07-09-81

CORPORATION

RECEIVED
SEP 1 1981
Pesticide-
[illegible]

August 28, 1981

COPY

~~Mr. Paul Barger~~
~~Maintenance Administrator~~
~~Dallas Independent School District~~
~~2538 South Ervay Street~~
~~Dallas, Texas~~

~~Dear Mr. Barger:~~

This letter will confirm completion of our soil removal and replacement activities at the Jose Navarro School, 3530 Kingbridge.

On Saturday, August 15, 1981, our contractor removed soil to a depth of six inches in an area around the swing set measuring approximately ten feet by thirty feet. Earlier samples had indicated that soil in that location might contain higher than normal levels of lead. One and a half truckloads of soil were removed. On Tuesday, August 18, 1981, two truckloads of soil were brought in and used to replace the soil removed on August 15. The replacement soil, which was tested and found not to contain significant quantities of lead or other contaminants, was packed to a depth and degree of firmness at least as great as the removed soil. Mr. Ray Lovejoy of the Dallas Independent School District was present during both the removal and replacement phases of the operation, and expressed his approval of the work performed.

Thank you for your cooperation in this matter.

Very truly yours,

Gerald A. Dumas

Gerald A. Dumas
Manager
Technical Services Department

GAD/wlf

cc: Dr. Norman Dyer ✓
Environmental Protection Agency

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ROBERT M. COHAN
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RESIDENT ATTORNEYS

RECEIVED
AUG 28 1981
Pesticides Branch
Dallas, Texas

August 27, 1981

CERTIFIED MAIL NO. 259439
RETURN RECEIPT REQUESTED

Dr. R. D. Gray
Chairman of the Board
Tabernacle Day Care Center
3403 McBroom
Dallas, Texas 75212

Dear Dr. Gray:

Several weeks ago I wrote to you requesting an opportunity for my client, RSR Corporation, and myself to meet with you to discuss the possibility of removing and replacing top soil at the Tabernacle Day Care Center. In the event you might have misplaced my earlier letter, I am enclosing another copy herewith. I am disappointed that I have received no response from you to that letter and no response from you or Dr. Reagans or anyone else at Tabernacle Day Care Center to the numerous phone calls that RSR personnel and I have made.

Although, as I indicated in my earlier letter, no health hazard has been found, the Environmental Protection Agency (EPA) believes that chronic exposure to high soil lead levels can increase the danger of high blood lead levels in children. Therefore, RSR renews its offer to Tabernacle Day Care Center to resample, and if necessary to replace, top soil in play areas where there is substantial risk of the soil being ingested by children. As stated in our earlier letter, this would be done without charge to the Tabernacle Day Care Center.

With the exception of the Tabernacle Day Care Center, RSR has now completed the initial phase of its soil resampling and replacement program. If you wish to have the Tabernacle Day Care Center included in that program, it is essential that you contact me within two weeks from the date of this letter. If I have not heard from you by that time, we must assume that the Tabernacle Day Care Center has no interest in having RSR's assistance in this matter.

Thank you very much for your cooperation.

Sincerely,

/S/

Robert M. Cohan
Attorney for RSR Corporation

RMC:mc
Enclosure

cc: ✓Dr. Norman Dyer
Environmental Protection Agency

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WASHINGTON OFFICE
1300 NINETEENTH STREET, N. W.
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(202) 828-1200

July 27, 1981

Dr. R. D. Gray
Chairman of the Board
Tabernacle Day Care Center
3403 McBroom
Dallas, Texas 75212

Dear Dr. Gray:

Pursuant to my telephone conversation with Mrs. Reagans last Friday, I am writing to request an early opportunity for my client, RSR Corporation, and myself to meet with you to discuss the possibility of removing and replacing some exposed top soil at the Tabernacle Day Care Center.

A study recently released by the Environmental Protection Agency (EPA) indicates that soil at some tested locations near Dallas lead plants contains higher than normal concentrations of lead. Tabernacle Day Care Center is one of those locations. Although no health hazard has been found, EPA considers the high lead levels to be an environmental concern. The source of the lead has not beem established. Nevertheless, RSR Corporation has advised EPA that, as a voluntary community service function, it will undertake to replace soil identified in the recent study as containing lead in excess of 1000 parts per million and located in high child-activity areas in elementary schools, playgrounds and day care centers.

We would anticipate removing and replacing only a couple of inches of top soil and only in play areas not covered with grass or other vegetation -- i.e., those areas in which there is the greatest risk of soil being ingested by children. Because RSR Corporation would be performing this service for the benefit of the community and without charge to the Tabernacle Day Care Center, we would require written consent and a waiver of liability from you for personal injury or property damage that might be attributed to RSR's presence at the Tabernacle Day Care Center or its soil-removal activity.

R
JUL 28 1981
Pesticides B[illegible]
Dallas, Texas

Please call me at your earliest convenience so that we may make appropriate arrangements. We hope to have all soil removal activities completed within the next couple of weeks. Thank you very much for your cooperation and assistance.

Sincerely,

Robert M. Cohan
Attorney for RSR Corporation

RMC:mc

cc: ✓Dr. Norman Dyer
Environmental Protection Agency

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

November 2, 1981

11/6/81 C.
CA
D
Dyer

OFFICE OF
PESTICIDES AND TOXIC SUBSTANCES

MEMORANDUM

SUBJECT: Status Report of Lead/Smelters Study Group

FROM: Walter W. Kovalick, Jr.
Director
Integration Staff

Walter W. Kovalick Jr

TO: Al Davis
Director
Air and Hazardous Materials Division

I am pleased to transmit to you the attached "Status Report of Lead/Smelters Study Group: November 2, 1981." This is the product of our ad-hoc work group which was appointed on October 6, 1981, by Dr. John W. Hernandez, Jr., Deputy Administrator of EPA.

We will contact you soon concerning your collaboration on the necessary additional analyses.

Attachment

STATUS REPORT OF
LEAD/SMELTERS STUDY GROUP

November 2, 1981

Interim Statement on Blood Lead and The Relationship of Soil to Blood Lead.

BACKGROUND

On October 6, 1981, EPA Deputy Administrator, Dr. John Hernandez, appointed a special study team to review the available data related to heavy metal contamination and possible health risks at the several smelters which EPA had studied across the country. The formation of this group implements a concept by which EPA will evaluate potential environmental health problems identified in research studies in a scientific and expeditious way.

The Lead/Smelters Study Group is divided into two teams--one of which is focusing on the environmental health and exposure aspects of the sites. The other team will develop, if necessary, the various action plans available for whatever remedial action may be necessary. Such plans will be developed and implemented in close concert with state and local governments.

The priority consideration for the Study Group was the review of the data associated with lead contamination in the Dallas area around smelter sites. The first task was for the Study Group to review the scientific literature related to blood lead levels as indicators of human health risks. Also, the group evaluated the literature related to the influence of soil contamination on blood levels. Following are the results of this review.

HEALTH ASPECTS OF LEAD

All people have lead in their body at some level (NAS, 1972). Elevated blood levels of lead are associated with adverse health effects, and there seems to be a continuum of induced changes that is related to blood levels (Chisolm & Barltrop, 1979; Needleman & Landrigan, 1981). The public health community has identified a level of concern by screening blood specimens from preschool children for elevated lead levels and biochemical alteration (Chisolm, 1977; Fielding & Russo, 1977). These measures can be used as indicators of excess lead exposure. As blood lead in preschool children extends above 30 ug/100 ml and is accompanied by erythrocyte protoporphyrin (EP) levels above 50 ug/100 ml, the Centers for Disease Control (CDC, 1978), recognize the need for some medical follow-up and environmental intervention. This is because of the fact that blood lead above 30 ug /100 ml in the presence of elevated EP is indicative of likely exposure which, if continued, may increase the body lead burden to levels associated with the induction of increasingly more serious health effects. The Agency recognized the CDC level of concern in setting the ambient air standard (USEPA, 1977). EPA acknowledges the desirability of identification of cases of undue lead absorption and determination of the impact of other continuing environmental lead exposures under its purview. Reducing such exposures is expected to lessen the likelihood of developing the medical problems outlined by CDC and others (Zarkowski, 1976).

SOURCES OF LEAD EXPOSURE

There are multiple sources of lead exposure to humans (Mahaffey, 1978). Foods, including home and commercially grown fruits and vegetables, canned items and other food sources constitute one major route of lead intake. The dusting and flaking of lead based paint from painted interior and exterior surfaces of buildings and dwellings constitute another significant source. Poorly lead-glazed ceramic pottery may also contribute to lead exposure, as will the consumption of improperly distilled, illegal liquor (i.e., moonshine)(Mahaffey & Goyer, 1974). Certain occupations are associated with lead exposures and, at times, lead can be carried home on contaminated clothing of lead workers (Baker et al., 1976; Rice et al., 1978). In addition, the air, water and soil constitute the pathways of environmental exposure of greatest concern to EPA.

Analyses of lead exposure problems have indicated multiple sources of exposure which probably contribute to the body level of lead (Landrigan et al., 1975; Angle & McIntire, 1979). Attempts to allocate the contributions of the various factors require careful inquiry and analysis of each potential source. In addition, relationships among environmental factors complicate such analyses. For instance, lead in the air is often related to that in the soil, that in house dust and that in the body of humans (Landrigan et al., 1975; Yankel et al., 1977; Angle & McIntire, 1979). Thus, in cases of elevated blood lead, there is the need for information on all sources of lead and the extent to which each may contribute to the body load (NAS, 1980).

Past inquiries on the part of the Agency as to the result of environmental exposures to lead via air (USEPA, 1977) and water (USEPA, 1980; Thomas et al., 1979) found a predictable relationship between lead in these environmental media and entry of lead into the body of exposed people. It was realized that increasing the body burden of lead above that commonly observed increased the chances of potential risk. Thus, to help minimize risks to health, the Agency developed regulations to decrease exposures to lead from ambient air and from water sources.

Recognizing that soil may represent still another source of exposure to lead, EPA has reviewed pertinent literature, and the following represents the Agency's initial position on the potential effects of elevated soil levels of lead. It also describes the approach EPA will use in evaluating possible sites of lead contamination.

SOIL LEAD EXPOSURES

The concentration of lead in soils is highly variable and is affected by several parameters (USEPA, 1977). These factors include airborne exposure from automobile exhausts and industrial sources, the dusting and flaking of lead paint from painted structures, and water runoff. Relationships have been noted between levels of lead in the soil and proximity to lead sources. Thus, soil lead levels decrease as a function of distance from lead industries (Roberts et al., 1974; Schmitt et al., 1979), heavily traveled roadways (Caprio et al., 1974) and structures covered with lead based paint (Bertinuson & Clark, 1973; Ter Haar & Aronow, 1974; Jordan & Hogan, 1975). The mobility of lead in soil is affected by many variables such as pH, soil type, composition, and organic content, all of which make it difficult to predict the movement of lead (Bolt & Bruggenwert, 1976; USEPA, 1976; USEPA, 1979). Soil lead can also redistribute into the air as particulates, migrate into water and move about the soil surface or be carried into homes and other structures as dusts.

Lead in soil is believed to be one of the sources of lead in humans. A limited amount of lead in soil may enter people through adsorption to or translocation into edible plants or inhalation of soil lead particles re-entrained into air. Of more concern is the evidence that indicates that soil and dust can be ingested especially by small children. Scientific evidence is accumulating that indicates a relationship between soil levels and measures of lead in blood (Vostal et al., 1974; Charney et al., 1980; Brunekreef et al., 1981), but the Agency finds that the exact nature of the association and EPA's ability to quantify it has not been fully established. Due to the interrelationship between soil concentrations with those in air and dusts and the finding that multiple media exposures may contribute to body lead levels (e.g., housedust, air, paint), it is difficult to isolate that portion of the body lead level which may be due to contamination of soil (Galke et al., 1975; Yankel et al., 1977). The Agency's previous analysis of the literature, as part of the development of regulations to implement the Clean Air Act, concluded that an association exists between soil and blood lead levels in cases where soil levels were high (EPA, 1977).

The ingestion of soil by small children follows from the observation that they tend to mouth items they handle. This activity can transfer small amounts of soil from their hands and articles to their mouths (Sayre et al., 1974; Lepow et al., 1975; Duggan & Williams, 1977; Roels et al., 1980). In addition, some youngsters eat larger quantities of certain nonfood items (pica) including soil (see Pueschel et al., 1977-78). Small children also absorb lead from the gastrointestinal tract more readily than do adults (Ziegler et al., 1978). The personal habits of older children and adults are much less likely to result in excessive exposure through contaminated soil (Baker et al., 1977; Roels et al., 1980).

Even with the information to date, there are numerous questions concerning the magnitude of the effects that soil lead may have on the body lead load of children. Examples of these questions include the frequency of mouthing of objects, the amount of material transferred from objects or hands to mouth, the extent of pica among children and the amount of material ingested, the chemical form of lead in the soil, the size of the transferred particles, and the extent of absorption of lead from various types of particles. Given the positive indications to date, but the uncertainties involved in the relationship between soil and blood lead, the National Academy of Sciences (NAS, 1980) has recommended that more research is needed to quantify the exposures that occur.

At present time the Agency concludes that it cannot with great certainty use soil levels to predict blood levels in exposed humans with precision or confidence. There is a recognition by the Agency, however, that soil lead levels can be useful in outlining geographical areas around sources of potential lead contamination where children can and should be surveyed for undue lead absorption.

SUMMARY AND AGENCY PLAN

EPA realizes the need to limit excessive exposures to lead in the environment. The Agency does not have a standard for levels of lead in soil, but it does recognize that research to date has demonstrated that soil is one of the exposure routes contributing to the lead burden in humans, especially young children. At this time the Agency does not believe that soil levels can be used to predict quantitatively blood levels in humans, but that elevated soil levels can be used to identify areas around lead sources that require further follow-up. As defined by the public health community, elevated blood lead and protoporphyrin screening levels among preschool children can be used to help determine cases of undue lead absorption.

In Dallas, the following is planned. EPA has already conducted a study of soil lead levels at selected sites around the smelters and battery plant in addition to some reference areas. The City is conducting a voluntary blood sampling program to gather information on the lead status of participants. The Agency and CDC will review these data to determine the adequacy of sampling, handling, and analysis of specimens and to measure the statistical strength of any association between soil levels and blood values. From this review, a determination will be made of the need for further environmental and biological follow-up. It is estimated that this preliminary assessment will be completed and further evaluation needs, if any, outlined within

12 weeks of receipt of all blood sample data. Various potential sources of lead exposure may need to be investigated including ambient air, drinking water, and lead based paint, as well as further soil sampling. After careful internal and external peer review of all information and follow-up studies and analyses, the Agency will make a determination of the need for mitigative action. Concentrations of lead in air and drinking water that are in excess of 1.5 ug/m^3 and 50 ug/l, respectively, can be addressed through existing regulations.

Similar strategies can then be adopted for other potential sources of lead contamination other than in the Dallas area. Soil, water and air near industrial plants can be evaluated for lead levels. Elevations in soil lead levels can be used to demarcate geographical areas for further investigation. If blood values of concern are linked to specific sources of contamination, the Agency will consider mitigative action. Excess lead concentrations in water and air can be handled through existing EPA regulations.

Questions and Answers

1. Q. How many lead smelters are there in the U.S.?

A. There are 55 secondary lead smelters, 17 of which are not currently operating. In addition, there are 7 primary lead smelters.

There are also many other possible sources of lead emissions which may lead to environmental problems. These include other non-ferrous metal smelters such as copper and zinc smelters and many other operations which use lead.

2. Q. Where are the smelters located?

A. They are located in 26 different states.

3. Q. What is the other half of the team doing regarding Dallas?

A. The available data for soil lead concentrations for the Dallas sites are being evaluated. Sampling methods, accuracy of analysis, and the relationship of lead concentration in the soil to distance from the smelter is being reviewed. Work is underway to determine what, if any additional data will be required to evaluate their potential routes of exposure such as air, groundwater, surface water, and house dust. Available groundwater data is being checked for the presence of lead. Ambient air data to measure compliance with air regulations is being evaluated with the state and local authorities. Available remedial action plans are being evaluated for use in the event that a need for action in any of these media should be determined.

4. Q. When will the Study Group report on the other five sites.

A. The Study Group will not review the other five sites until peer review of the ORD report including the sampling techniques, analytical methods, quality assurance, and analysis of data is complete. At that point, we will review other sites in a similar manner.

5. Q. What are the most useful screening tests for lead?

A. The Centers for Disease Control have found that the most useful exams are blood analyses of lead and protoporphyrin. The lead level gives some indication of absorption, while the protoporphyrin level is a measure of inhibition of heme synthesis, a necessary component of hemoglobin of red blood cells.

6. Q. Does an elevated blood Pb level from a screening test mean my child has Pb poisoning?

A. No, screening tests are not diagnostic and can only be used to identify persons for follow-up evaluation. Cases of confirmed blood elevation should be seen by a physician for more detailed evaluation.

7. Q. Are blood screening tests for acute or chronic health effects?

A. The aim of lead screening program is to detect promptly the children with undue Pb absorption so that appropriate medical and environmental actions can hopefully be taken before the onset of symptoms. Whether the undue Pb absorption occurred rapidly or slowly, a risk exists and actions must be taken to protect the child.

8. Q. If blood lead values are elevated around the smelters in Dallas where soil lead is high, does that implicate soil as the problem?

A. No. First, EPA has not yet evaluated the blood lead study conducted by the City of Dallas, so we are not yet certain about any elevated blood levels in the area. Second, EPA has not independently monitored ambient air and drinking water, and we may not have an inclusive view of soil levels in the area. Still other factors would need to be investigated, such as the occurrence of lead base paint in homes and other structures, and the extent of use of earthenware in homes. After an investigation of these sources, a better evaluation of the role of each factor can be made.

9. Q. What is the value of screening for lead in human hair and nails?

A. Hair and nails are unsuitable specimens for lead screening purposes due to the inaccuracy of the quantitative analysis of Pb in these specimens. The surfaces of these specimens are frequently contaminated with Pb from external sources, such as cosmetics and dust; therefore, the surfaces must be appropriately cleaned before the analysis. Using the presently available cleaning procedures, Pb is not only removed from the surface, but is also leached out of the specimen, and the measured Pb content of the specimen is too low.

10. Q. Is tooth analysis a valuable screening tool for potential adverse effects of lead in humans?

A. Recent studies involving Pb analysis of deciduous teeth have provided significant and promising results; hoever, additional development and experience are needed before tooth Pb analysis could be considered appropriate for screening purposes. Particular areas that need further study are identifying the effects of surface contamination in analytical results, determining the best practicable sampling procedures, and correlating tooth Pb levels with potential risks.

11. Q. Aren't there potential risks to the fetus from maternal lead exposures?

A. Early this century it was recognized that pregnant women employed in the lead industry had an increased frequency of miscarriages and stillbirths. Steps were taken at that time to limit the exposure to women during pregnancy. Since then little information has been accumulated on risks to the unborn. One might assume that the fetus is as susceptible as small children. Therefore, exposures to lead during pregnancy should be limited; exposed women should contact their physicians.

(3)

12. Q. When will the Study Group complete the review of the Dallas blood data?

A. From the time of receipt of the last blood sampling results it will take about 12 weeks for the Agency to reach a definitive position. This is due to the number of activities that must be carried out in that time period including the coding of blood sample data into a computer, performing summary statistics, determining the adequacy of sample preparation, handling and analysis, measuring associations between soil and blood values, developing follow-up plans and having them evaluated by scientists both inside and outside the Agency.

13. Q. Who is on the Study Team?

A. The Team is an ad-hoc group composed of Agency scientists, engineers, and others who are experienced in hazard assessment, exposure analysis, environmental clean-up. It's membership includes both those with general hazard and exposure evaluation experience as well as those in EPA's regulatory programs that have dealt with lead. In the short time available, [illegible] also consulted with CDC and Dr. Julian Chisholm of Johns Hopkins University and Rufus Chaney of USDA. Other outside scientific input will be consulted as we move to evaluate of the blood lead data from the City of Dallas.

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STATUS REPORT ON THE LEAD/CADMIUM STUDY CONDUCTED IN DALLAS AND A SCENARIO OF POSSIBLE ACTIONS

EPA, REGION VI

JUNE 29, 1981

UNITED TES ENVIRONMENTAL PROTECTI GENCY

DATE

UBJECT Status Report on the Dallas Lead/Cadmium Study

FROM Frances E. Phillips
Acting Regional Administrator, Region VI

TO Dr. John Hernandez
Deputy Administrator

I have attached a status report on the Lead and Cadmium Study conducted in Dallas, including a scenario of recommended actions for addressing elevated blood and/or soil lead levels in the study area.

Region VI is recommending cleanup by the companies in areas where a statistically significant percent of the blood lead levels exceed 30ug/100ml (level recognized by the Center for Disease Control (CDC) as the threshold level above which clinically significant adverse effects are noted in exposed children). In areas where a significant percent of blood lead levels are 20-30ug/100ml, the Region would recommend that the city of Dallas conduct a public education program and encourage the city and companies to monitor the blood lead levels of children residing in areas with high soil lead levels. In areas where there is not a significant percent of blood lead levels above 20ug/100ml, the Region would not recommend any further action.

If an area must be cleaned up, the Region has included a rough cost estimate for preventive measures ranging from $150,000 for the immediate area around the facilities (a circle around each plant 1/4 mile in radius) to $450,000 for the downwind areas within one to two miles of the facilities.

Attachments

TABLE OF CONTENTS

I. Historical Perspective
  A. Background
  B. Rationale for the Study Approach and Selection of Areas
II. Dallas Study
  A. Background
    1. Purpose
    2. Strategy
      a. Contract
      b. Time Period
      c. Sample Protocol
    3. Results
  B. Present Status
    1. Discussions with City, State, and Federal Officials
    2. Discussion with Companies
      a. RSR
      b. General Battery Corporation (Owners of Dixie Metals)
      c. N. L. Industries
  C. Future Plans
III. Health Effects and Significance of Environmental Exposure
  A. Action Blood Level
  B. Action Soil Level
  C. Environmental Pathways

IV. Mechanisms for Remedial Action

- A. Voluntary Action by Industry
- B. Action by State and Local Governments
- C. EPA Action
    - 1. Section 7003 of the Resource Conservation and Recovery Act
    - 2. Section 303 of the Clean Air Act
    - 3. Superfund
    - 4. Common Law of Nuisance

V. Policy Ramifications

- A. Setting an Action Level
- B. Precedent for Cleaning Up Areas Other than Industrial
- C. Setting a Precedent for Demonstrating Adverse Health Effects

I. Historical Perspective

A. Background

The Toxic Substances Control Act (TSCA) was signed into law on October 11, 1976. This law is designed to fill the gaps between the other environmental and occupational laws and to enable the Federal Government to control unreasonable risks to human health and the environment caused by chemical substances.

Because TSCA can only be used when other laws cannot adequately address a chemical problem, there must be coordination among the various programs in EPA with other Federal agencies. Thus, Region VI established a Toxic Substances Coordinating Committee (TSCC) to ensure a multimedia and interdivisional approach. This committee was given the responsibility for preparing an integrated toxic substances strategy. This strategy was prepared to coordinate intergovernmental, multimedia chemical control in Region VI to address potential public health problems. Emphasis was placed on the most significant potential problems and on regulatory consistency to ensure efficient use of Federal/State/local resources.

After completing the Regional Integrated Strategy, the committee reviewed data on all chemicals produced or processed in Region VI in significant quantities and selected six groups of chemicals as regional priority chemicals. The committee based the selection of these chemicals on the magnitude of production, the known multimedia environmental problems (based on available monitoring), toxicity, and existing controls. The chemicals selected in priority order are as follows:

lead
cadmium
brominated compounds
benzene and derivatives
phenols
butadiene and derivatives

Lead and cadmium were selected as the two top priority regional chemicals because of the vast amount of data which showed widespread environmental contamination in Region VI and exposure of children residing in contaminated areas. Further, according to the Center for Disease Control (CDC), during fiscal year 1979 more than 32,500 children in the United States showed symptoms of lead toxicity, compared to 25,800 during fiscal 1978. The CDC stated that an improved screening program accounted for some of the increase, but also estimated that 1 percent of America's children between the ages of one and five suffer from excess lead absorption.

The Region focused its attention on soil and dust as sources of the lead, because EPA's present lead control programs in air and water appear to adequately address these potential problem sources. A recent exposure model had identified soil as a major contributor to urban childhood exposures (See Attachment 1, Exposure Model). Also, none of the existing media programs addressed soil as a major source of exposure for sensitive subgroups of the population (children and pregnant women).

Soil and dust data from previous studies had shown contamination ranging from several hundred parts per million (ppm) to several thousand ppm in several areas in Region VI: Bartlesville, Oklahoma, and Dallas, El Paso, and Corpus Christi, Texas (Attachment 1). Blood lead levels of numerous children living in some of the areas exceeded the 30 micrograms per 100 milliliters (ug/100ml) level, which the CDC recognizes as the threshold level above which clinically significant adverse effects are noted. These areas of known environmental contamination were located in communities surrounding nonferrous smelters.

A 1972 Dallas study consisted of blood samples obtained from children living around both RSR and Dixie Metals/N.L. Industries. These data showed an average blood lead level of 30ug/100ml around Dixie Metals/N.L. Industries and 22ug/100ml around RSR. As a result of this study, the companies were required to install emission control devices. N.L. Industries closed in 1979, and Dixie Metals did not install emission controls which operated properly until 1979. RSR also had new controls placed on its plant in 1979.

With this background, TSCC developed a chemical work plan which consisted of a two phased approach for addressing potential lead and cadmium contamination in areas surrounding smelter activities. This first phase was prepared as a screening program to identify areas with elevated lead and cadmium levels in soil, dust, and water which could present a potential health problem. If phase I showed elevated soil levels, a second phase was planned to clearly delineate the extent of lead and cadmium contamination and to determine the relationship in children between elevated blood lead levels and soil levels. If a relationship between environmental contamination and the exposure of children was established in phase II, follow-up action was planned to reduce the exposure.

B. Rationale for the Study Approach and Selection of Areas

The Region VI priority locations for study were around secondary lead smelters located near residential areas because these smelters contribute to air, soil, dust, water, and vegetation exposure pathways for small children. The result of the soil and dust lead contamination is continuous exposure through resuspension, even after the lead facility has ceased operation or installed emission control equipment. This exposure to lead is the cumulative result of exposure from all media, i.e., air, water, food, soil, dust, etc.

Since EPA did not have an existing program that provided a mechanism for cleanup of existing contaminated soil sites, EPA initiated a program of intense State and local awareness to generate support for the study and the possibility of future cleanup of these sites. State or local action was deemed necessary because: (1) this was considered a problem best addressed by local or state government; and (2) this plan was developed before the Agency initiated the abandoned site program or had gained much experience in enforcement of the Resource Conservation and Recovery Act (RCRA), or Superfund.

Region VI contracted with local universities and government agencies to conduct the soil, dust, and water studies. By contracting with local organizations, the Region hoped to gain more local support as well as greater acceptance of the study results and recommended corrective measures.

Based on the location of historical point source lead emissions, the studies were conducted in Baton Rouge and Shreveport, Louisiana; Bartlesville and Oklahoma City, Oklahoma; and Dallas, Houston, and Frisco, Texas. The studies in Baton Rouge and Shreveport have been completed but did not show any significant elevation in lead/cadmium levels. Studies are currently being conducted in Frisco, Houston, and at a site in Dallas. Studies were recently completed around three areas in Dallas and one area in Bartlesville; a limited study has also been completed at Oklahoma City.

II. Dallas Study

A. Background

1. Purpose

The Dallas study was initiated to determine if there was a potential lead exposure problem around several sources in the city. Dallas has three lead smelters: RSR Corporation, Dixie Metals (General Battery owns Dixie Metals), and N.L. Industries (closed in 1979). The city also has two battery plants: General Battery Corporation and ESB, Inc. The first study involved the three smelters and General Battery. ESB, Inc. is currently being studied.

2. Strategy

a. Contract

A procurement announcement on the study was mailed to local universities in the Dallas area; the contract was awarded to the University of Texas at Arlington on September 29, 1980, for $9,999.

b. Time Period

The contractor took samples in October, November, and December of 1980. The analyses were completed in January 1981. The report was provided to EPA on February 24, 1981.

c. Sample Protocol

The contractor collected soil, dust, and water samples from houses and other locations within a two mile radius of the potential emission sources and in a control area. These samples were prepared for analysis using the standard preparation techniques and analysed for lead and cadmium using an atomic absorption spectrometer (AAS). A flame AAS was used for soil and dust analyses and a flameless AAS for water analyses.

Quality assurance procedures included the use of reagent blanks, the replication of ten percent of all AAS readings, and the use of standards every tenth sample. In addition, all glassware and other items used in the analysis procedures were acid-washed in concentrated nitric acid before use.

Ten houses were sampled around each facility and the control area. Outside the house, four to six soil samples were collected: two from the front yard, two from the back yard, and two from any garden that was present. Each soil sample consisted of a core sample three inches deep. Inside each house, the contractor (1) took a dust wipe from a window sill and the top of the refrigerator; (2) placed a twenty-eight day dust tray on the refrigerator or another item in the house where the tray would be left undisturbed; and (3) took a sample of both cold and hot tap water.

In addition, the contractor took four soil samples from ten other locations around each facility and three other locations at the control area. These other locations consisted of elementary school yards, parks or playgrounds, and daycare centers.

3. Results

Cadmium soil levels found throughout Dallas were not high enough to be of major concern. The average levels found in this study ranged from 0.25 ppm for the control area to 2.05 ppm around RSR Corporation (See Attachment 2). The Office of Toxic Substances uses 3.0 ppm cadmium as a warning level for further study. The hazard criteria is exceeded when at least ten percent of the samples exceed 3.0 ppm. The levels of cadmium found around the RSR facility approached this level, with nine percent of the samples exceeding 3.0 ppm cadmium and ten percent of the locations where samples were taken having an average value above 3.0 ppm cadmium. However, it should be noted that the use of 3.0 ppm cadmium as a warning level is based solely on the normal cadmium soil levels present in the United States. The 3 ppm level is an estimate used by the Field Studies Branch in the Office of Toxic Substances; however, this level has no scientific support.

Soil lead levels were elevated around all of the facilities studied. The average values of lead found in this study were 54 ppm for the control area, 131 ppm around General Battery, 494 ppm around RSR, and 1254 (670-716) ppm around Dixie Metals/N.L. Industries (Dixie Metals and N.L. Industries are located across the street from each other.) The Dixie Metals/N.L. Industries area had two soil samples with exceptionally high levels of lead (24,000 and 25,000 ppm). If these two values are dropped, the average lead value at Dixie/N.L. Industries drops to 670 ppm. If the Region replaces these two values with 5200 ppm lead (the total for the other two soil samples in that yard), the average value is 716 ppm. The yard with the two values above 20,000 ppm has been resampled and two other areas in the front yard were found to contain 2253 and 2187 ppm lead. Therefore, the high values are only isolated problems which are probably the result of contamination by the home owner and not plant emissions. The contractor informed EPA that the area where these two original samples came from was black and that the resident may have drained the oil from his car in these areas.

High levels of lead are present in the soil around both the RSR and Dixie Metals/N.L. Industries facilities. Twenty-five percent of the soil samples around the RSR facility and thirty percent of the soil samples around the Dixie Metals/N.L. Industries facility are above 1000 ppm lead, which is the level used as a hazard warning for further study by the Office of Toxic Substances. A table showing the soil lead concentrations is provided as Attachment 3. The contractor is resampling other areas to determine the soil lead concentration in the first inch of soil versus the first three inches. This information will reveal whether the soil contamination problem identified is isolated in the surface soil. If most of the lead contamination is in the top inch of soil, then there would be additional locations in each area with soil contamination above 1000 ppm lead, areas which would be readily accessible to children.

The tap water results verify Federal, State, and local agency reports that there are no problems with lead and cadmium contaminated drinking water in Dallas. In addition, the dust sampling results did not show a major problem. The dust levels in houses in the study areas were higher than those found in the control area, but they were not high enough to cause any health problems. This conclusion is based upon information found in the Air Quality Criteria for Lead which was published by EPA in December 1977. It should be noted that it rained on eight different days during the sampling period; therefore, lead dust levels could be higher during the dry summer months when Dallas receives almost no rain.

B. Present Status

1. Discussion with the City, State, and Federal Officials

After receiving the results from the study, we notified Congressmen in an April 7, 1981, letter. These letters included the average values of lead found around each facility and the future plans for a blood lead study to determine if a health risk exists. (Copies of these letters are enclosed as Attachment 4). These letters were mailed to Martin Frost, Phil Gramm, Jim Mattox, and Jim Collins of the House of Representatives, and John Tower and Lloyd Bentsen of the Senate.

On April 16, 1981, EPA held a meeting with State and local officials to discuss the results from the study. The meeting was attended by representatives from the Texas Department of Water Resources, the Texas Department of Park and Wildlife, the Texas Air Control Board, the Texas Health Department, the City Health Department, the Mayor's Office, and the Southwestern Medical School. The direction of the follow-up action was assumed by the city of Dallas and EPA. The city agreed to take the lead on resolving the health problem with EPA focusing on the environmental problem. EPA asked the city to provide the Region with blood lead data obtained through the Early Periodic Screening and Diagnostic Program (EPSDT), on children living around the smelters in Dallas to determine if there was a correlation between elevated soil levels found in the EPA study and the city's blood lead levels.

A follow-up meeting was held with city officials on April 27 to review the EPSDT blood lead data. This data showed that 44 percent of the 27 children who were reported as having high blood lead levels lived within two miles of one of the smelters.

On May 18, EPA met with the Assistant City Manager, Mr. Levi Davis. At the meeting, the City Health Department and the Director of the Children and Youth Project in West Dallas agreed to give EPA a map showing where blood lead samples had been taken around the smelters and a copy of the blood lead levels found. The Assistant City Manager agreed that if these data were insufficient to determine if there was a blood lead problem in the study areas, the city would conduct a blood screening program in the areas of concern.

The maps on the blood lead levels provided by the city after this meeting were again inconclusive. Only a small number of children were checked around the smelters and the exact blood lead concentrations and length of residency were not given. (The results were only reported as above or below 30ug/100ml). As a result, the city has agreed to conduct a blood lead screening program through its neighborhood clinics which are located near the lead facilities. The screening program will be conducted June 29-July 13, 1981.

2. Discussion with Companies

In designing the lead/cadmium strategy, the Region planned to work with the companies in a voluntary mode to facilitate resolving any environmental problems identified. However, if an agreement on an appropriate plan of action could not be obtained, the local/State agencies or Region VI would also be in a position to initiate an enforcement action to require the cleanup of contaminated areas. In our initial discussions with the companies, the Region selected 1000 ppm lead as a level which would require corrective measures, such as removal. This level was based on studies which found 1000 ppm lead in soil as unacceptable (See Attachment 5). This concept has been revised, however, to require cleanup only if a health problem is found, that is, elevated blood lead levels corresponding to the CDC health threshold level.

The Region received several Freedom of Information (FOI) and news media requests before it had a chance to discuss the report with the city or companies involved. Responses to these requests were delayed until the Region could meet with the city and the companies and develop action plans.

Vic Argento, the contractor who conducted the study, contacted RSR Corporation and Dixie Metals regarding the results of the study. This was done without the Region's knowledge or permission. Subsequently, RSR contacted Region VI concerning a meeting to discuss the results of the study and offer its assistance in correcting any problem.

a. RSR Corporation

The first meeting with RSR was held on April 21, 1981. The Region discussed the results from the study and indicated the 1000 ppm lead level as a level of concern. At this meeting, RSR confirmed that it would clean up these contaminated areas and presented a written statement that it would cooperate with EPA.

The Region met with RSR again on May 28 to review its plans developed in accordance with the initial meeting and also to review a draft EPA press release. The preparation of the press release was prompted by information from a reporter that The Dallas Morning News was going to publish an article the next day on the lead contamination problem in Dallas. The Region, however, reached an agreement with the editor to delay its publication until June 1 to allow time for the Region to meet with the remaining companies and then with the media. The report and news release were given to the media on June 1, 1981.

At the meeting with RSR, the company agreed to clean up any soil areas over 1000 ppm lead and to support financially a blood lead study in the vicinity of its facility. In addition, RSR made comments on the content of the press release and provided us with a revised version on May 30.

A third meeting was held on June 10, 1981. In addition to RSR, General Battery Corporation (Dixie Metals) was also present at this meeting. Joe Carra, Office of Toxic Substances, and Steve Williams, a contractor to EPA from Research Triangle Institute, were also present to discuss the appropriate protocols to be used in the soil and blood studies. RSR presented a plan for conducting additional soil sampling, a blood study, and cleanup of areas with above 1000 ppm lead. The Region, however, advised the companies that our position had been modified and that the Region would not require immediate cleanup of those areas containing 1000 ppm lead in soil. EPA would determine which areas needed cleanup only after additional studies to correlate elevated soil with elevated blood levels (i.e., threat to public health).

At the conclusion of the meeting, the companies were asked to devise a study design to identify the contaminated soil areas and the blood lead levels of residents around its facility. Both companies agreed to contact the region in approximately two weeks. RSR's study would be contracted out to a local physician and the University of Texas at Arlington.

On June 26, the Region met with RSR to: clarify the phase II study protocol; discuss EPA's position on RSR's voluntary decision to begin immediate cleanup of community (i.e., daycare center, schools) sites with soil levels above 1000 ppm; and request EPA policy on action level and cleanup measures.

b. General Battery Corporation (Owners of Dixie Metals)

An initial meeting was held with General Battery Corporation (Dixie Metals) on April 23, 1981. The results of the study were discussed at the meeting, along with our concern for those areas with high levels of lead (1000-4000 ppm). General Battery was not as cooperative as RSR and wanted to point out that N.L. Industries had operated a lead facility across the street for a number of years (it was closed in 1979). The company was very concerned about the release of the study results and possible lawsuits which might result. General Battery wanted to meet with us again before the release of the report and mentioned the possibility of buying some of the homes if a health problem was identified.

On May 28, a second meeting was held with General Battery Corporation (Dixie Metals) to discuss its plans and the content of a press release. General Battery had a number of attorneys and a consultant, Dr. S. Lerner from the University of Cincinnati, present at the meeting. The company did not agree with an action level for lead in soil, unless it could be linked to a health problem. The literature references addressing 1000 ppm as a problem and a draft press release were reviewed. General Battery agreed to conduct a blood study in the vicinity of the Dixie facility. On May 30, General Battery provided the Region with comments on the draft press release.

On June 10, at the request of the companies a joint meeting was held with both RSR and General Battery Corporation. As discussed previously, the design protocols for the blood and soil lead studies were discussed. General Battery agreed to conduct the blood lead study and to provide the Region a copy of their design for review within two weeks. In addition to the items previously mentioned by RSR, the issue of automobile lead was brought up be General Battery. The Region discussed the existence of an isotope analysis to distinguish between lead from automobile emissions and smelters. General Battery agreed to contact the Region regarding their study design in approximately two weeks. A subsequent call by General Battery requested a delay until the week of July 13.

c. N. L. Industries

Only one meeting has been held with N. L. Industries. This meeting was held on Sunday, May 31, 1981, to discuss the results of the study and the press release. The lawyers at this meeting were not in a position to make policy decisions and therefore did not make any commitment. However, in a follow-up letter dated June 15, 1981, N. L. Industries has agreed to work with General Battery Corporation in financing the proposed blood and soil studies the Region requested.

C. Future Plans

Both RSR and General Battery will be contacting us in July to provide their plans for a blood screening and soil sampling program for our review. The data provided from this study will be made available to the city of Dallas Health Department. Any children who are found to have elevated blood lead levels will be referred to Children's Hospital in Dallas.

If a health problem is found (i.e., blood lead levels above 30ug/100ml) preventive measures would be taken such as soil removal, paving, or acidic fertilization. If the companies do not voluntarily cooperate in this effort, it may become necessary to initiate enforcement actions to require these preventive measures.

After the results from the studies by the companies become available, working through the city, the residents in the affected areas will be briefed on study results and any planned follow-up actions. Subsequently, a press conference will be held to discuss the findings and plans for any needed cleanup actions.

III. Health Effects and Significance of Environmental Exposures

A. Action Blood Level

Blood lead is the most widely used indicator of recent lead exposure. It is regarded as a reasonable surrogate for the biological response associated with lead absorption. The intent of any health strategy for lead should be to lower the blood lead levels of the exposed population. Specifically, the intent should be to reduce the blood lead levels of those subgroups of the general population who have a greater risk of lead exposure (children) or who are more sensitive to lead than the rest of the population (fetuses and children). Since the American Pediatrics Association and the CDC have suggested that 30ug/100 ml blood lead is that threshold above which clinically significant adverse effects are noted in exposed children, it is prudent to address individuals whose blood lead levels are 30ug/100ml or higher. However, it should be emphasized that blood lead is only a good marker of recent exposure, and even though exposure is excessive, blood lead levels could return to normal. Errors are likely to occur, therefore, if blood lead is used to classify subjects after exposure has ceased. Dr. Herbert L. Needleman has shown that lead levels in teeth are a better indicator of chronic exposure.

The neurotoxic (nerve damage) properties of lead at high dose levels are well known and not a subject of general controversy. A source of considerable debate, however, is whether or not blood lead levels below those associated with obvious symptoms have adverse effects on the brain. Because the symptoms of milder lead intoxication are not dramatic and may therefore evade precise identification, many efforts have been made to determine whether lesser levels of lead are associated with undetected neuropsychologic impairment (learning disorders).

Several studies (See Attachment 5, Reference 13) show that a current or past history of moderate elevations of blood lead is an important factor in producing significant impairment in cognitive, verbal, perceptual, and fine motor skills when compared to a control group with levels of lead below 30ug/100ml. Needleman demonstrated that children with high lead levels in the dentine of the teeth scored significantly lower on the Wechsler Intelligence Scale for Children than those with low dentine levels.

B. Action Soil Level

Numerous studies have shown correlations between elevated soil and dust (air emissions) lead levels and increased blood lead levels in children living in those environments (See Attachment 5, References 2, 4, 5, and 10). These studies link 1000 ppm lead in soil as the cutoff level that should be considered unacceptable and at which removal or containment is required. All of the above studies except Reference 4 show a direct correlation between 1000 ppm lead in soil and increased blood lead levels. No correlation was noted in Reference 4 possibly because the area of concern was under development (park) and exposure was minimal.

In the Silver Valley Study (See Attachment 5, Reference 1) blood lead levels were found to be most influenced by five variables: ambient air lead, soil lead, age of child, dustiness of the home, and occupational status of parents. Blood lead levels were dramatically reduced, in a five year period, in El Paso, Texas, as a result of lowered levels of lead in air, dust, and soil initiated by engineering improvements on the smelter there (See Attachment 5, Reference 12). In this follow-up study it was stated that almost all children living in El Paso within 0.8Km of the smelting plant were evacuated to more distant, less heavily contaminated areas. Other studies (See Attachment 5, References 3, 6, 7, and 8) have shown a significant health hazard from elevated dust, soil, and air lead levels.

C. Environmental Pathways

Because the toxicological effects associated with lead exposure can be considered cumulative, it is imperative to define and quantify all major sources of human lead exposure. Ambient air, food, and drinking water are the major sources of exposure for adults. Soil and dust, through common hand-to-mouth activity, and various lead-containing materials, through pica, are significant additional sources of exposure to children. Inhalation and ingestion of lead containing substances by adults and children appear to be the predominant routes of exposure.

Humans are exposed to lead and lead-containing compounds through the various environmental pathways illustrated in Attachment 6. The Region's study focused on tap water, dust, and soil as possible sources of lead exposure because State and local records show the companies are in compliance with lead air emission standards.

IV. Mechanisms for Remedial Action

The following actions could be employed to remove or clean up contaminated soil.

A. Voluntary Action by Industry

Voluntary action by industry will expedite the identification of the extent of soil lead contamination and any blood lead elevation in Dallas. Such cooperative efforts save significant amounts of time and resources.

B. Action by State and Local Governments

If the industries choose not to proceed with voluntary remedial measures, Texas or the city of Dallas could proceed if they possess legal authority, technical expertise, and sufficient resources to effect the necessary remedial measures.

The Region has every indication that the city of Dallas will take care of any identified public health problems. It is possible that the Texas Air Control Board would take enforcement action, if it could be demonstrated that existing air emissions are causing the health problem. The Texas Department of Water Resources has not taken an active role, since the soil contamination does not involve classical disposal. The Texas Department of Health has deferred to the city for action. Without industry cooperation, State/city lead is the preferred course of action.

C. EPA Action

As a last resort, and in the face of a public health problem, EPA could proceed under several statutes.

1. Section 7003 of the Resource Conservation and Recovery Act

If handling, storage, treatment, transportation, or disposal of any solid or hazardous waste is creating an imminent and substantial endangerment to health or the environment, the Administrator may commence a civil action to require remedial action. This section only requires that a potential health risk exists, not that actual harm has occurred.

2. Section 303 of the Clean Air Act

If an air pollution source is presenting an imminent and substantial endangerment to the health of persons, the Administrator may commence civil action to take necessary remedial measures. While similar to actions taken pursuant to Section 7003 of RCRA, actions taken under this emergency provision of the Clean Air Act are authorized only when the health of persons is jeopardized, not when the environment alone is threatened.

In addition, action under Section 303 of the Clean Air Act can be taken only if the State and local authorities have not abated the pollution. Also, this section of the Act could only be used if there is a current air emission problem, which is not the case in Dallas according to State/local officials.

3. Superfund

While it appears that Superfund could be used to further identify and remedy elevated soil lead and blood lead levels found near the Dallas smelters, this option presents serious practical impediments. Notably, Superfund is untested, and this is not a classical abandoned site.

4. Common Law of Nuisance

In spite of the Supreme Court's recent decision in the City of Milwaukee vs. Illinois, an action based on the common law of nuisance should not be entirely discounted. Action brought as a result of the Dallas Lead Study could be distinguished from the above decision by the lack of a comprehensive regulatory scheme addressing lead contamination. EPA's authority to bring suit under the common law of nuisance remains somewhat unclear.

V. Policy Ramifications

A. Setting an Action Level

If the Region chooses a level of soil lead, such as 1000 ppm, which corresponds to elevated blood lead levels (those greater than 30ug/100ml) as an action level requiring cleanup, it may be interpreted as a national action level. Such a precedent could require that other Regions clean up lead-contaminated areas at the same level, even though this is a site-specific policy dependent upon several factors: soil PH, vegetation, rainfall, etc. In another area, the action level could be higher or lower, depending on these factors. This policy could also impact enforcement actions in other areas and Regions under both RCRA and Superfund.

B. Precedent for Cleaning Up Areas Other Than Industrial

If a cleanup level is set for lead, do we only clean up contaminated areas resulting from smelters, or do we also include areas contaminated by automobile emissions? In Dallas, a traffic study was conducted by Southwest Research Institute under contract to EPA in 1978 to determine the relationship between lead soil concentrations and traffic density (See Attachment 7). This study did not find any neighborhood soil levels above 1000 ppm lead and only three samples above 500 ppm lead. The average level of lead found in this study was 128 ppm. However, the city of Dallas collected soil samples along the borders of major freeways and found several values above 1000 ppm lead. These soil samples, however, were not in residential areas; therefore, the exposure of children would be precluded. The 1978 Dallas traffic study and our lead study focused on residential areas.

C. Setting a Precedent for Demonstrating Adverse Health Effects

Under RCRA and the Clean Water Act, legal actions are taken against industries solely on the basis that a pollutant is present at a certain level without proof that a health problem exists. If we delay cleanup until proof of a health problem we may be setting a national precedent contrary to our current enforcement policies.

VI. Scenarios of Possible Preventive Measures

A. Discussion of the Approach

To simulate the areas needing cleanup, the study areas were stratified into smaller sections using concentric circles and/or areas focusing on the downwind direction from each facility location. The areas were stratified into 1/4 mile sections extending out from the point source. In reality, however, actual boundaries would be based on the company's sampling program.

A preliminary land survey was conducted and city maps (census and planning) were used to identify the mix of land usage in each section (i.e., single family and multifamily residences, school playgrounds, parks, industrial areas, vacant lots) and an estimate of the population impacted. Using this information, a scenario was developed which indicates the types of preventive measures that would be recommended given a certain combination of factors (See Charts 1 and 2). These charts indicate preventive measures to be taken when the blood lead levels in a particular area exceed 30ug/100ml. The measures are typical, but the actual measures would be based on a site by site analysis and include a consideration of the probability of childhood exposures. This approach is necessary because the extent of cover (grass) or containment (pavement) will influence the degree of exposure of children through common hand-to-mouth activities in play areas. For those yards with grass (thick turf), a recommendation could be made for the companies to use an acidic fertilizer to reduce the surface soil lead levels, followed by retesting to determine the reduction in soil lead levels. If the industrial areas are paved, further action would not be necessary. In the floodplain, no action would be deemed necessary, unless the area was used as a park.

1. Blood Lead Levels greater than 30ug/100ml

If the blood levels are above 30ug/100ml, Region VI would recommend taking some preventive measures to reduce the potential exposure of the children. Depending on the site, the degree of blood lead elevations, and the soil lead concentrations, the measures could consist of the following:

a. Encourage seeding and use of acidic fertilizers to stimulate plant (grass) growth in dusty play areas and to mobilize lead.

b. Turn over top layers of soil and mix them with layers underneath to dilute the lead concentration in the top layer, then seed and fertilize to increase grass growth.

c. Place at least four inches of new top soil over contaminated dusty yards and stimulate grass growth.

d. Contain soil with asphalt or pavement.

e. Remove contaminated soil to appropriate depth and replace with clean top soil.

f. Move residences from contaminated area (used most frequently in other cities for residences close to the smelter complex).

2. Blood Lead Level 20-30ug/100ml

On the other hand, if the percent of blood lead levels between 20-30ug/100ml is statistically significant in the study areas, the city would be asked to distribute a list of preventive measures, including the following, through neighborhood clinics, local schools, daycare centers, and community meetings.

a. Control dust inside the home by damp-mopping floors and frequent dusting/cleaning (using oil base polish or damp cloth).

b. Control dust and dirt exposure outside of the home by establishing and maintaining grass cover and watering during dry periods.

c. Wash thoroughly before eating fruits and vegetables grown in home gardens within the contaminated area.

d. Since small children are quite sensitive to metals, take special precautions to ensure that children do not eat dirt or put articles covered with dirt in their mouths. A sand box or other play areas should be supplied for small children who normally play outside in the dirt.

e. Reduce exposure through good personal hygiene (washing hands before eating, frequent bathing and washing hair).

f. Provide adequate supply of calcium in children's diets, since children with calcium deficiency will be affected more quickly by metals than those with sufficient calcium. Milk is an excellent source of calcium.

g. Recommend annual screening of blood lead levels through existing city screening program/Children's Hospital.

h. Encourage seeding and use of acidic or acid phosphate fertilizers to stimulate the growth of grass and the release of the bound lead.

3. Blood Lead Levels less than 20ug/100ml

Finally, if all of the blood lead levels are below 20ug/100ml or only an insignificant number of blood lead levels are above 20ug/100ml, the Region would not recommend any action.

B. Cost Estimate for Removal of Soils

A rough estimate has been made for soil removal and replacement of an individual lot. This estimate can be multiplied by the anticipated number of lots to be cleaned. The cost for removal of soil in a park or playground area can be approximated by dividing the square feet in the playground area by 3750 and multiplying by the cost of cleanup of an individual lot.

The rough cost estimate is based on removal of the first four inches of top soil on a 3750 square foot lot, replacing with four inches of new top soil, and fertilizing and seeding. That cost would average approximately $600/lot.

The cost to transport the material to a disposal site would be dependent on the distance of a haul from the place of removal to an approved disposal site. The cost for the soil removed from a typical lot would be $250 for a disposal site within forty miles of the contaminated areas which is the distance to the nearest hazardous waste disposal site which has complied with the RCRA permit requirements.

Costs for removal, replacement, and transport of contaminated soil to a disposal site would be approximately $850/lot. The disposal fee charged by the landfill accepting the soil has not been included in the estimate. This charge would be largely based on the quantity of soil to be disposed and its characteristics (i.e., whether or not it meets the RCRA criteria for classification as a hazardous waste). There might be a possibility of negotiating a one time disposal of the soil in a local landfill if it could be demonstrated that there would be no hazard created and EPA/State agencies agreed to such an arrangement. If the soil is not a hazardous waste, it could be disposed in a local sanitary landfill. In either case, the disposal fee would probably be approximately $40-60 per lot.

All efforts must be made to control dust generation during these operations. Dust control procedures, such as soil wetting during excavation and use of covered trucks during transport will be required. Heavy equipment operators should be required to wear respirators.

A more complete and accurate cost estimate can be made when the disposal site and the number of yards to be cleaned up are determined.

BLOOD LEAD LEVELS

		30 µg/100 ml or greater	20 to 30 µg/100 ml	Less than 20 µg/100 ml
SOIL LEAD LEVELS	High (i.e., 1000 ppm or greater)	Site clean up Dallas Public Education Program	Dallas Public Education Program	No Action
	Medium (i.e., 500 ppm)	Site clean up Dallas Public Education Program	Dallas Public Education Program	No Action
	Low (i.e., 100 ppm)	Site clean up Dallas Public Education Program Additional Pathway Study Encouraged	Dallas Public Education Program	No Action

1. All scenarios include a joint city/EPA press release and city sponsored public meetings in the study areas.

2. The city of Dallas will refer children with blood levels of 30 ug/100 ml or above to Children's Hospital for medical attention.

RSR DOWNWIND STUDY AREA

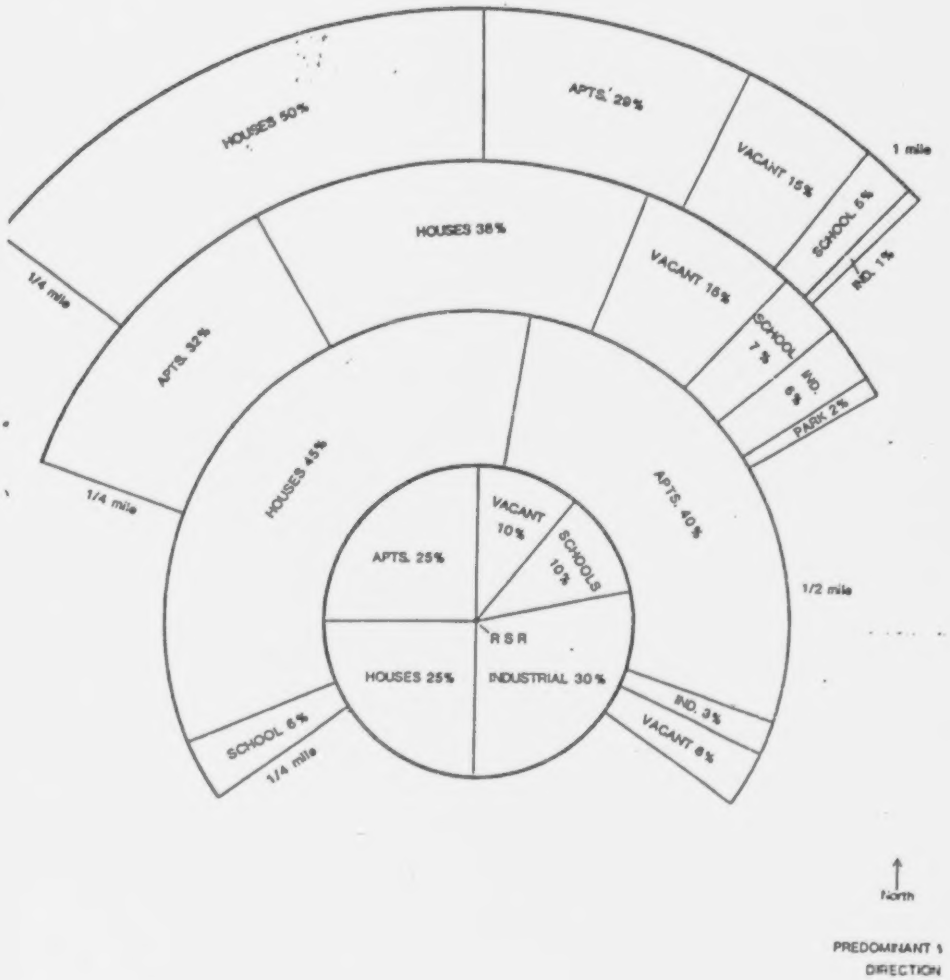

HOUSES 50%
APTS. 29%
VACANT 15%
SCHOOL 5%
IND. 1%
1 mile
1/4 mile
HOUSES 38%
APTS. 32%
VACANT 15%
SCHOOL 7%
IND. 6%
PARK 2%
1/4 mile
HOUSES 45%
APTS. 40%
1/2 mile
IND. 3%
VACANT 6%
SCHOOL 6%
1/4 mile
APTS. 25%
VACANT 10%
SCHOOLS 10%
HOUSES 25%
INDUSTRIAL 30%
RSR
North
PREDOMINANT
DIRECTION

CHART 1, Page 1

RSR

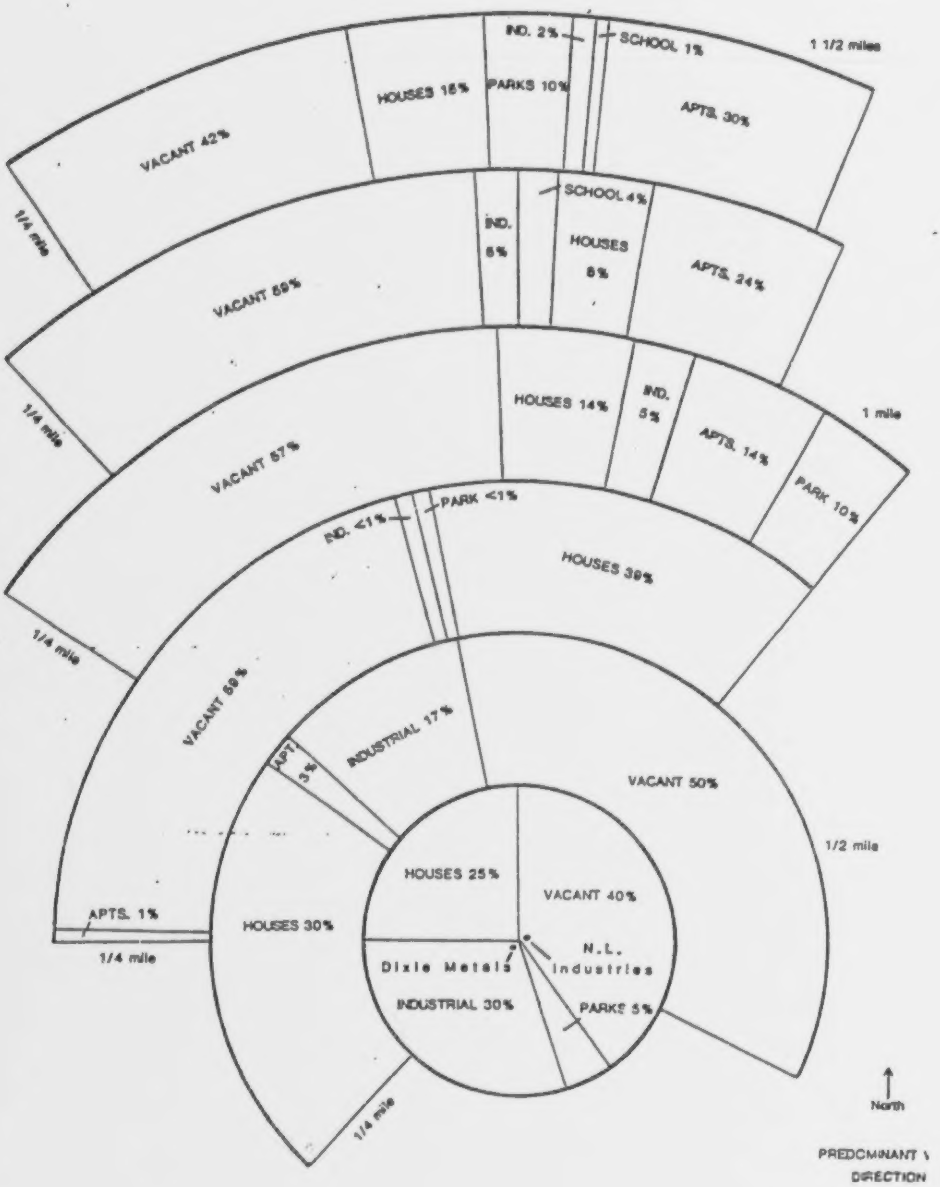
Blood Lead Level (ug/dl)	Size of Contaminated Area and Estimated Population	Land (Site) Usage and (Number)	Per Cent Usage	Preventive Measures	Who Will Perform	Estimated Cost
Greater Than 30	1/4 Mile Circle	76 SF	25%	Remove/Pave/Seed/Fertilizer	Company	30,000
		196 MF	25%	Remove/Pave/Seed/Fertilizer	Company	15,000
	SF 549	1 Schools	10%	Remove/Pave/Fertilizer	Company/City	4,000
	MF 1098	- Industrial	30%	Pave/No Action	Company/Owner	5,000
		- Parks	0%	---	---	
		- Vacant lots or under-developed	10%	Seed/Fertilizer	Company/City/Owner	2,000
						56,000
	1/4-1/2 Mile Segment 210 degrees of circle	260 SF	45%	Remove/Pave/Seed/Fertilizer	Company	22,000
		225 MF	40%	Remove/Pave/Seed/Fertilizer	Company	20,000
	SF 1098	3 Schools	6%	Remove/Pave/Fertilizer	Company/City	14,000
	MF 1098	- Industrial	3%	Pave/No Action	Company/Owner	2,000
		- Parks	0%	---	---	
		- Vacant lots	6%	Seed/Fertilizer	Company/City/Owner	1,000
						59,000 (115,000)
	1/2-3/4 Mile Segment 140 degrees of circle	317 SF	38%	Remove/Pave/Seed/Fertilizer	Company	27,000
		250 MF	32%	Remove/Pave/Seed/Fertilizer	Company	22,000
	SF 549	2 Schools	7%	Remove/Pave/Fertilizer	Company/City	8,000
	MF 1098	- Industrial	6%	Pave/No Action	Company/Owner	3,000
		1 Parks	2%	Fertilizer/Pave/Remove	Company/City	3,500
		- Vacant lots	15%	Seed/Fertilizer	Company/City/Owner	2,000
						65,500 (180,500)

CHART 1, Page 2

RSR

Blood Lead Level (ug/dl)	Size of Contaminated Area and Estimated Population	Land (Site) Usage and (Number)	Per Cent Usage	Preventive Measures	Who Will Perform	Estimated Cost
Greater Than 30	3/4-1 Mile Segment 90 degrees of circle	411 SF	50%	Remove/Pave/Seed/Fertilizer	Company	27,500
		150 MF	29%	Remove/Pave/Seed/Fertilizer	Company	14,000
	SF 1000	2 School	5%	Remove/Pave/Fertilizer	Company/City	8,000
	MF 300	- Industrial	1%	Pave/No Action	Company/Owner	1,000
		- Parks	0%	---	---	
		Vacant lots	15%	Seed/Fertilizer	Company/City/Owner	2,000
						52,500
						(233,000)

DOWNWIND STUDY AREA

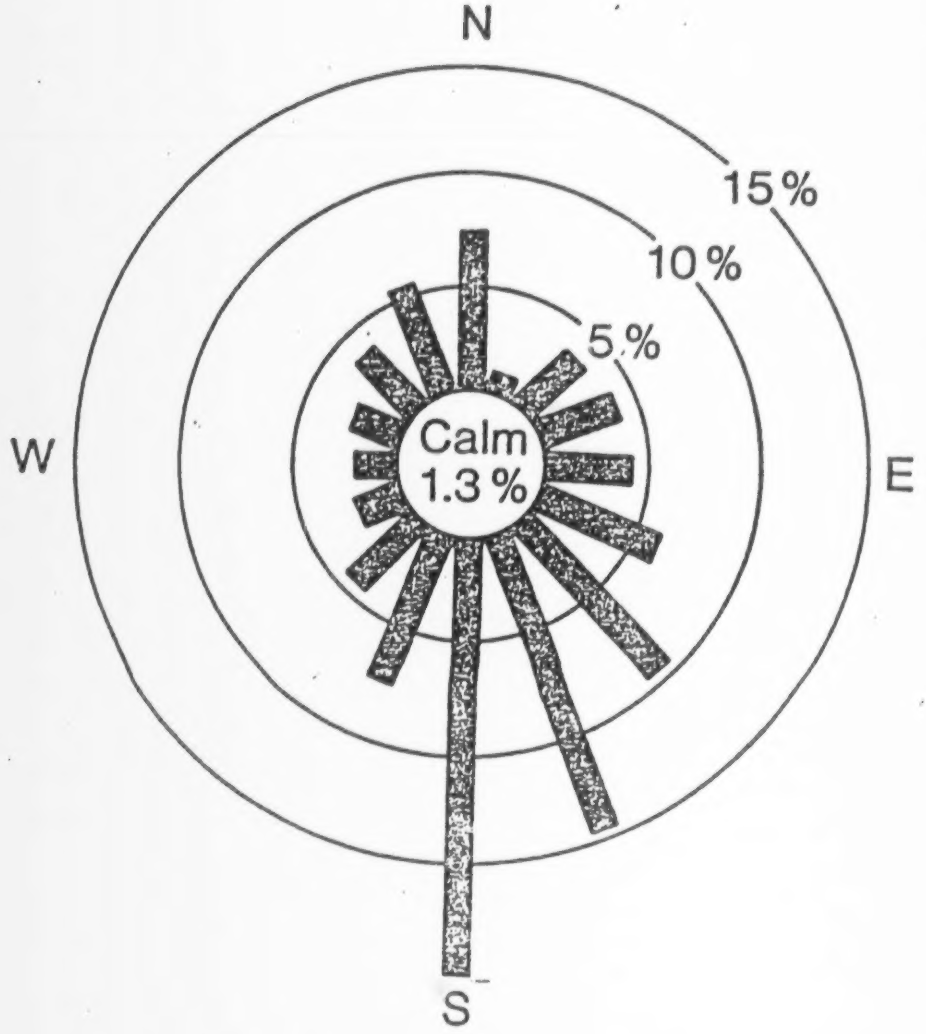

VACANT 42%
HOUSES 15%
PARKS 10%
IND. 2%
SCHOOL 1%
APTS. 30%
1 1/2 miles
1/4 mile
VACANT 59%
IND. 6%
SCHOOL 4%
HOUSES 8%
APTS. 24%
1/4 mile
VACANT 57%
HOUSES 14%
IND. 5%
APTS. 14%
PARK 10%
1 mile
1/4 mile
IND. <1%
PARK <1%
HOUSES 39%
VACANT 59%
APTS. 1%
1/4 mile
INDUSTRIAL 17%
APT. 3%
VACANT 50%
1/2 mile
HOUSES 30%
HOUSES 25%
VACANT 40%
Dixie Metals
N.L. Industries
INDUSTRIAL 30%
PARKS 5%
1/4 mile
North
PREDOMINANT
DIRECTION

Blood Lead Level (ug/dl)	Size of Contaminated Area and Estimated Population	Land (Site) Usage and (Number)	Per Cent Usage	Preventive Measures	Who Will Perform	Estimated Cost
Greater Than 30	1/4 Mile Circle	106 S-F	25%	Remove/Pave	Company	70-80,000
		0 M-F	0%	---	---	
	SF 320	0 Schools	0%	---	---	
	MF 0	Industrial	30%	Pave/No Action	Company/Owner	5,000
		Parks	5%	Fertilizer/Pave/Remove	Company/City	7,000
		Vacant lots (Floodplain)	40%	Remove/Seed/Fertilizer (No Action)	Company/City/Owner	12,000
						94-104,000
	1/4-1/2 Mile Segment 210 degrees of circle	204 S-F	43%	Remove/Fertilizer/Seed/Pave	Company	35,000
		- M-F	5%	Remove/Pave/Fertilizer	Company	6,000
	SF 600	0 Schools		---	---	
	MF 120	- Industrial	25%	Pave/No Action	Company/Owner	9,000
		0 Parks	0%	---	---	
		Vacant (Floodplain)	27%	Seed/Fertilizer (No Action)	Company/City/Owner	2,000
						52,000 (150,000 Tot)
	1/2-3/4 Mile Segment 95 degrees of circle	433 S-F	39%	Seed/Fertilizer/Remove	Company	15,000
		- M-F	1%	Pave/Seed/Fertilizer	Company	500
	SF 1300	- Schools	0%	---	---	
	MF 70	- Industrial	1%	Pave/No Action	Company/Owner	500
		2 Parks	1%	Fertilizer/Pave/Remove	Company/City	1,000
		- Vacant (Floodplain)	59%	Seed/Fertilizer (No Action)	Company/City/Owner	5,000
						22,000 (172,000)

CHART 2, Page 2

Dixie Metals/N.L. Industries

lood ead evel ug/dl)	Size of Contaminated Area and Estimated Population	Land (Site) Usage and (Number)	Per Cent Usage	Preventive Measures	Who Will Perform	Estimated Cost
reater Than 30	3/4-1 Mile Segment 90 degrees of circle	140 S-F	14%	Remove/Seed/Fertilizer/Pave	Company	4,500
		- M-F	14%	Pave/Seed/Fertilizer	Company	2,000
	SF 420	0 Schools	0%	---	---	
	MF 400	- Industrial	5%	Pave/No Action	Company/Owner	1,000
		1 Parks	10%	Fertilizer/Pave/Remove	Company/City	5,000
		- Vacant (Floodplain)	57%	Seed/Fertilizer (No Action)	Company/City/Owner	500
						13,000 (185,000)
	1-1 1/4 Mile Segment 60 degrees of circle	15-25 SF	8%	Remove/Seed/Fertilizer/Pave	Company	6,500
		- MF	24%	Seed/Fertilizer/Pave	Company	5,000
	SF 80	2 Schools	4%	Remove/Pave/Fertilizer	Company/City	6,000
	MF 650	- Industrial	5%	Pave/No Action	Company/Owner	2,000
		1 Parks	0%	---	---	
		- Vacant (Floodplain)	59%	Seed/Fertilizer (No Action)	Company/City/Owner	1,000
						20,500 (205,000)
	1¼-1½ Mile Segment 55 degrees of circle	15-25 SF	15%	Remove/Seed/Fertilizer/Pave	Company	6,000
		- MF	30%	Seed/Fertilizer/Pave	Company	5,500
	SF 70	1 Schools	1%	Remove/Pave/Fertilizer	Company/City	2,000
	MF 750	- Industrial	2%	Pave/No Action	Company/Owner	500
		3 Parks	10%	Fertilizer/Pave/Remove	Company/City	5,000
		- Vacant (Floodplain)	42%	Seed/Fertilizer (No Action)	Company/City/Owner	1,000
						20,000 (225,500)



AVERAGE WIND DIRECTION
DALLAS LOVE FIELD 1961–1973

ATTACHMENT 1

Studies on Lead Facilities in Region VI/Exposure Model

Bartlesville, Oklahoma

1. Arsenic, lead, and cadmium absorption levels were determined in 1,774 children 1-5 years old living in 19 USA towns with primary nonferrous metal smelters. Results were compared with data on 258 children of the same age in 3 communities without smelters.

   The highest mean blood cadmium (.50ug/100ml), hair cadmium (9.14ug/gm), and blood lead (28.60ug/100ml) levels encountered in the entire study were in the children tested in Bartlesville, and the mean hair lead levels there were higher than in any other zinc smelter town.

   Baker, Edward, Hayes, Carl G. and Landrigan, Phillip. A Nationwide Survey of Heavy Metal Absorption in Children Living Near Primary Copper, Lead, and Zinc Smelters. Unpublished Paper supported by HEW, EPA, PHS, and CDC, 1975.

2. As the result of an in-home study prompted by a citizen complaint, analysis of certain soil samples showed very large enrichments of zinc, lead, cadmium and copper in Bartlesville. Lead levels ranged from 62 ppm to 1,300 ppm where as the cadmium values ranged from less than 1 to 500 ppm at one site. Other soil samples collected downwind of the National Zinc plant contained metal concentrations averaging 22,500 ppm for zinc, 1650 ppm for lead, 350 ppm for cadmium and 510 ppm for copper.

   The Environmental Input of Emissions from the National Zinc Company in Bartlesville, Oklahoma. Environmental Protection Agency, Office of Enforcement (NEIC). EPA-330/2-78-003.

3. Four smelter communities were identified as sampling sites for this study. Two zinc smelters (Bartlesville, OK and Palmerton, PA) and two copper smelters (Azo, AZ and Anaconda, MT) were chosen; a zinc processing community Corpus Christi, TX) was selected as a pre-test site. Bartlesville had the highest zinc levels in household soil (1-5 years old play area). Lead concentrations in household soil, dust, and food ingested by study participants were also highest over all sites. A corresponding maximum level of blood lead was noted for the 1-5 year old group. Cadmium levels in soil, food ingested by study participants, and dust were highest at this site and were reflected in maximum cadmium hair concentrations for both the 1-5 year olds and all study subjects.

   RTI - Epidemiologic Study Conducted in Populations Living Around-Nonferrous Smelters. Final Report, May 15, 1981 (Not available for public release). Study was under the direction of Dr. Carl G. Hayes. Health Effects Research Laboratory RTI.

4. Results from a Regional study showed 1420 ppm lead and 190 ppm cadmium in animal feed lot samples. While Roof Sweeping showed concentrations of 3450 ppm lead and 476 ppm cadmium.

   EPA, Ada, Oklahoma - Analysis of Heavy Metals in Soil from a feed lot between National Zinc and two schools north of the site and roof sweepings from several homes to make a composite sample. July 1977.

5. In March 1979, the Regional Office collected soil samples from fourteen elementary schools in Bartlesville for analysis of heavy metals. Lead levels at Lincoln Elementary were significantly elevated (Surface-1705 ppm and Sub-Surface 797 ppm). Cadmium levels at Jane Phillips and Lincoln Elementary Schools were significantly high (Surface 68.7 ppm and Sub-Surface 15.95 ppm at Jane Phillips; Surface 58.6 ppm and Sub-Surface 24.7 ppm at Lincoln).

   EPA, Ada, Oklahoma - March 1979.

Corpus Christi, Texas

As mentioned before, four smelter communities were identified as sampling sites in the RTI study. Corpus Christi was selected as a pre-test site.

Based on the median concentrations of the samples collected at the five sites studied, Corpus Christi had the highest cadmium levels in drinking water (household and school) and maximum cadmium concentrations in blood for both 1-5 year olds and all study participants. In the absence of high environmental lead, Corpus Christi participants had the highest lead hair concentrations for both 1-5 year olds and all study subjects.

RTI - Epidemiologic Study Conducted in Populations Around-Nonferrous Smelters. Final Report May 15, 1981 (Not available for public release). Study was under the direction of Dr. Carl G. Hayes, Health Effects Research Laboratory, RTI.

Dallas, Texas

1. In July 1972, the Dallas City Health Department conducted a blood lead screening program for children in two areas of the city. The test area was located in the vicinity of Dixie Metals/N.L. Industries and the control area was located around RSR Corporation. The average blood levels found were 30ug/100ml in the test area and 22ug/100ml in the control area.

   W. C. Johnson, M.D. and James P. Luby, M.D. A Report on a Study to Determine the Blood Lead Levels in Dallas Children.

2. The Southwest Research Institute conducted a study in Dallas on the effect of Automobile Traffic on Blood Lead Levels. The objective of this research project was to characterize the absorption of lead by people of different age-sex groups exposed to automobile emissions of lead at traffic densities from less than 1000 cars per day. Examination of the relationship to traffic counts greater than 1000 to less than 25,000 cars per day with concentrations of lead in the blood of residents revealed no detectable relationship. No significant relationship was found between traffic counts and FEP, hematocrits, carbon monoxide levels in blood, and lead in hand-wipes.

   Southwest Research Institute, Epidemiologic Study of the Effect of Automobile Traffic on Blood Lead Levels. EPA Contract Number 68-02-2227 prepared by Dr. Warren Galke, Population Studies Division of RTI - January 1978.

El Paso, Texas

1. Blood lead levels were determined on a radom sample of persons in all age groups living near a lead-emitting smelter in El Paso, Texas. A blood lead level of greater than or equal to 40ug/100ml, which was considered indicative of undue lead absorption (this level was reduced to 30ug/100ml in 1975), was found in 53 percent of the children one to nine years old living within 1.6Km of the smelter and in 18 percent of those from 1.6 to 6.6Km; beyond that distance and in older persons levels were lower. Children in the first 1.6Km with blood levels greater than or equal to 40ug/100ml were exposed to thirty-one times as much lead in dust as children with lower blood values (6447 vs. 2067 ppm). There was also air-borne lead exposure (8 to 10ug/cubic meter, annual mean). Paint, water, food, and pottery were less important as sources.

   Landrigan, P. J., Gehlbach, Stephen, Rosenblum, Bernard, et.al. Epidemic Lead Absorption Near An Ore Smelter: The Role of Particulate Lead. New England J Med 292:123-129,1975).

2. Increased lead absorption was found in 1972 in 56 percent of 256 children ages 1 to 18 years old, who lived within 1.6Km of a lead-emitting ore smelter in El Paso, Texas. Engineering improvements have subsequently reduced emissions from the smelter, and levels of lead in air, dust, and soil have declined. To evaluate the impact of these environmental improvements on lead absorption, a follow-up study was conducted in 1977 of blood lead levels in 140 children ages 1 through 18 years old, who lived within 1.6Km of the smelter. Mean blood lead levels were found to have decreased from 41.4 to 17.7ug/100ml in children living within 0.8Km of the plant and from 31.2 to 20.2ug/100ml in children living at 0.8 to 1.6Km. This data argues for the feasibility of reducing children's lead absorption near primary lead smelters.

Exposure Model

Hand to mouth activity (e.g., thumb sucking and finger licking) resulting in the ingestion of soil and dust is a normal characteristic of children. Within a lead contaminated environment, hand-to-mouth activity and immature dietary habits (i.e., the retrieval of food from dusty surfaces or soil, and subsequent consumption), make soil and dust a major source of ingested lead for children.

Calculation Sequence in Determining Source Contribution Factors: Childhood Case

Source	X	Concentration	X	Consumption Rate	X	Absorption Rate	-	Daily Uptake in Children	Percent of Total Daily Uptake
Drinking Water		50 ug/l		1.4 liters per day		0.50		35.0 ug/day	5.8
Air		1.5 ug/m^3		4.7 m^3/day		0.40		2.8 ug/day	1
Food		93 ug/day		--		0.50		46.5 ug/day	7.7
Soil/ dust		11,000 ug/g		100 mg/day		0.30		330.0 ug/day	54.3
Paint		8,000 ug/g		142 ug/day		0.17		193 ug/day	31.8
						Total		607.3 ug/day	

Steven C. Orill, James J. Konz, et al, The Environmental Lead Problem: An Assessment of Lead in Drinking Water from a Multi-Media Perspective, 1979.

ATTACHMENT 2

CADMIUM LEVELS

Cd in Soil (ppm)	Dallas DMC 84	RSR 80	GBC 84	Ref. 54	Ref 44	Baton Rouge Daniel 49	Schuyk 37	Ethyl 46
0-1	28	25	71	54	42	33	3	12
1-2	32	34	9	0	1	1	22	13
2-3	18	14	3	0	0	6	7	18
3-4	3	1	0	0	1	9	5	2
4-10	3	5	1	0	0	0	0	1
>10	0	1	0	0	0	0	0	0
AVER.	1.50	2.05	0.58	0.25	0.43	1.03	1.72	1.91

ATTACHMENT 3

LEAD IN SOILS -- DALLAS TEXAS -- 1981 SURVEY

ppm of Lead	DIXIE METALS	RSR	GENERAL BATTERY	REFERENCE
0-49	4	4	15	44
50-99	22	10	29	8
100-499	28	42	37	2
500-999	5	5	2	0
1000	25	20	1	0
AVERAGE	1253.7(670-716)	494.1	131.3	54.0

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

DATE: April 5, 1983

SUBJECT: Speaking Points of Frances Phillips in Reference
to Dallas Lead Study

FROM: John A. West
John A. West
Pesticides & Toxic Substances Branch, 6AW-P

TO: Allyn M. Davis, Director
Air & Waste Management Division, 6AW

Thru: Norman E. Dyer, Chief Norman E Dyer
Pesticides & Toxic Substances Branch, 6AW-P

I have searched the Regional lead files relative to the Dallas Lead Studies and I have been unable to identify any speaking points used by Ms. Frances Phillips during early June of 1981 in a conversation with Dr. Hernandez in regard to the Dallas soil lead contamination problem.

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION VI
1201 ELM STREET
DALLAS, TEXAS 75270

March 29, 1983

Mr. Robert S. Prolman
Professional Staff Member
Subcommittee on Investigations
  and Oversight
Committee on Public Works
  and Transportation
U.S. House of Representative
B-376 Rayburn House Office Bldg.
Washington, D.C. 20515

Dear Sir:

In response to your request for documentation for certain statements made in reply to questions during the Hearing, I am submitting the following material:

I. In reference to the questions which were raised about the soil lead/blood lead relationship, I am submitting the following published scientific articles and a Federal Register Notice:

  A. Angle, Carol & McIntire, Matilda S., "Environmental Lead and Children: The OMAHA Study, Journal of Toxicology and Environmental Health, 5:855-870, 1979.

  B. Roels, Harry A., et al, Exposure to Lead by the oral and the Pulmonary Routes of Children Living in the Vicinity of a Primary Lead Smelter, Environmental Research, 22:81-94 (1980).

  C. Lepow, Martha L., et al, Investigations into Sources of Lead in the Environment of Urban Children, Environmental Research, 0, 415-426 (1975).

  D. Brunekreef, Bert, The Arnhem Lead Study, Environmental Research 25, 441-448 (1981).

  E. EPA National Ambient Air Quality Standard for Lead, page 42256, published in the Federal Register, Thursday, October 5, 1978 Part IV.

F. Angle, C.R. & McIntire, M.S., Children - The Barometer of Environmental Lead, accepted for Advances in pediatrics 1982 (Ann. Pub.) 10/10/81.

G. Roberts, T.M. and et al, Lead Contamination around secondary smelters: Estimation of Dispersal and Accumulation of Humans, Science, vol. 186.

H. Moore, Michael R., and et al, The Association Between Lead Concentrations in Teeth and Domestic Water Lead Concentrations, Clinica Chimica Acta, 87 (1978) 77-83.

II. In response to questions raised about biochemical and nervous systems effects of lead at blood levels below 30 ug/dl, I submit the following:

A. Piomelli, Sergio, et al, Threshold for Lead Damage to Heme Synthesis in Urban Children, Proceeding of the National Academy of Sciences, vol. 79, pp. 3335-3339, May 1982.

"Preliminary analysis suggested that" an exponential increase in the concentration of EP occurred after a threshold blood lead level of between 12 and 20 ug/dl was reached.

B. Benignus, V.A., et al, Effects of Age and Body Lead Burden on CNS Function in Yound Children. II. EEG Spectra, Electroencepholography and Clinical Neurophysiology, 1981, 52: 240-248.

"Increased blood lead generally produced" an increase in the relative amplitude of synchronized EEG between P3 and P4 in all frequency bands.

"This was true for blood lead levels well below 15 ug/dl among the lowest level of blood lead effects previously reported."

C. Landrigan, Philip J., et al, Neuroepidemiologic Evaluations of Children with Chronic Increased Lead Absorption, Low Level Lead Exposure: The Clinical Implications of Current Research, edited by H.L. Needleman, Raven Press (1980) 17-23.

"The clear implication to be drawn from these data is that lead exposure, particularly of children, ought to be reduced to the lowest limit possible.

D. Statement by Dr. Vernon N. Houk, Director, Center for Environmental Health, Centers for Disease Control, before the Subcommittee on Environment, Energy and Natural Resources, Committee on Government Operations, U.S. House of Representatives, Washington, D.C., April 14, 1982, page 3.

III. Several articles on soil lead clean-up levels.

IV. Finally material is provided regarding the QA/QC evaluation of the Dallas Lead soil sampling and Analysis procedures.

This information is submitted to the Subcommittee on Investigations and Oversight Committee on Public Works and Transportation, Hearings on Hazardous Waste Contamination of Water Resources, specifically the Dallas Lead Studies.

I respectfully request that this information be added to the record of my testimony.

Sincerely,

Norman E. Dyer

Norman E. Dyer

Accepted for
Advances in Pedia[illegible]
10/10/81

I. Soil Lead/Blood Lead Relationship

CHILDREN - THE BAROMETER OF ENVIRONMENTAL LEAD

CAROL R. ANGLE, M.D.

Department of Pediatrics
University of Nebraska Medical Center
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MATILDA S. McINTIRE, M.D.

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CHILDREN - THE BAROMETER OF ENVIRONMENTAL LEAD

The expanding definition of the new morbidity in pediatrics now encompasses health related environmental problems as well as the psychosocial domain. Pediatricians are increasingly concerned with the potential effects of prenatal and postnatal exposure to enviromental toxins. Some of these toxins, such as formaldehyde, once considered relatively innocuous, have more recently been shown to be responsible for significant symptoms at trace levels far below the previous threshold of detection. The halogenated hydrocarbons, particularly the polybrominated and polychlorinated biphenls (PBBs, PCBs) 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), are relative newcomers that have rapidly resulted in worldwide contamination of potentially high toxicity. At the far end of the time scale is lead, an enviromental contaminant for so many thousands of years that complex extrapolations are needed to define its natural levels. The cumulative knowledge of the environmental contamination with lead and its biologic consequences for children and adults as well as animals, wildfowl, and vegetation, provides an ecological case study in which the methods of analysis are relevant to the examination of other environmental toxins.

Lead poisoning has existed since antiquity with classical clinical symptoms correctly attributed to lead as early as the second century B.C.[1] It has, however, been only during the past two decades that the generalized risk from environmental contamination with lead has been recognized as a public health problem. Until the 1960's lead poisoning was primarily considered an episodic problem occurring in young children with pica, as well as being an occupational disease of lead and other industrial workers.

CHILDREN - THE BAROMETER OF ENVIRONMENTAL LEAD

The interaction of childhood lead poisoning with industrial sources was, however, well documented in many 19th century reports of the reproductive toxicity in women employed in the painting of pottery and the general ill health of children living in mountainous valleys where the meteorlogical conditions favored a constant fallout of lead dust from industrial sources.[2,3,4] As important as these epidemics were in identifying the risk of environmental lead intoxication, they represent the extremes in exposure resulting in severe lead poisoning with irreversible toxicity. A more basic question is whether the current levels of environmental lead pose a universal risk by elevation of the body burden of lead in the general population to the threshold of toxicity.

Historical Aspects

Paleoarcneology proposes that environmental contamination to lead began more than 9000 years ago with the hammering and use of native copper followed by cast copper objects about 7000 years ago.[5] (Fig.1) In the next 1000 years metallurgists recognized the metallic impurities of smelted copper and developed the tin or arsenic alloys of copper that introduced the bronze age.[6] About 6000 years ago, lead was probably first produced as a by-product of silver smelting. The discovery soon followed that a small amount of silver could be smelted from lead sulfide ore, with further purification of silver from the smelted lead-silver alloy by a second smelting process called cupellation. Until the Industrial Revolution, lead was primarily the residue of silver production with about 400 tons of lead oxide obtained for each ton of silver yielded by cupellation.

CHILDREN - THE BAROMETER OF ENVIRONMENTAL LEAD

The development of analytic methods to determine the purity of precious metals and the evolution of the concepts and tecnniques of weights and measures gave rise to the first widespread use of silver coinage during the classic Greek period.[7] Not surprisingly there were wide discrepancies in the production of lead throughout the Old World. Wealthy countries had silver mines; they also applied their ingenuity to the abundance of lead. By-product lead was used in building construction, pigments, cosmetics, ship sheathing, aqueducts and gutter systems. Both the Athenians and the Romans prepared a product called sapa by simmering sweet grape juice with spices and herbs in covered, pure lead kettles. Sapa, containing several thousand ppm lead, was added to green wine as a bactericide to prevent souring.[5] The widespread utilization of sapa is thought to be the principle reason why human bones from the time of the Roman empire have lead concentrations at least four times contemporary values. Whatever the contribution of epidemic lead poisoning to instances of insanity and sterility in the ruling class and thus to the downfall of the Roman empire, economists agree that exhaustion of the lead-silver mines was fiscally catastrophic to both Athens and Rome.[8] The health effects may have been beneficial. Lead smelting and refining had long been recognized as hazardous trades assigned to settlements of slave labor. When the mines were depleted, lead smelting became a local industry involving relatively few workers. It was not until the discovery of the New World and the exploitation of silver and lead deposits in Mexico and Peru that lead production again reached the level of the Roman empire. Patterson has proposed that the transfer of 40,000 tons of silver from the

New World was a major factor in the downfall of the Spanish economy by the suppression of local industry, heavy purchases of foreign goods, and a negative balance of trade.[5]

Despite the recognition of lead smelting as the cause of a clinical syndrome and the knowledge that lead was the major ingredient of sapa, the specific hazard of lead contamination in wine was not clearly recognized until the 17th century when laws were passed throughout Europe and North America prohibiting its use, as noted by Benjamin Franklin in his report of the risk of New England rum:[9]

> "The First Thing I remember of this kind was a general Discourse in Boston, when I was a Boy, of a Complaint from North Carolina against New England Rum, that it poisn'd their People, giving them the Dry Bellyach, with Loss of the Use of their Limbs. The Distilleries being examin'd on the occasion, it was found that several of them used Leaden Still-heads and Worms, and the Physicians were of Opinion, that the Mischief was occasioned by the Use of Lead. The Legislature of the Massachusetts thereupon pass'd an Act, prohibiting under severe Penalties the Use of such Still-heads and Worms thereafter. Inclos'd I send you a Copy of the Act. taken from my printed Law-Book."

The use of lead as a bactericide in the production of wine continued to be a common practice until Pasteur's discovery of bacteria and the introduction of sulfur dioxide.

With the Industrial Revolution lead entered the market as a primary product rather than a by-product of silver. The rapid increase was accompanied by a growing recognition of its risks to workers.[10] It was not until the 20th century that the protection of the lead workers first entered the public domain and the specific risks were documented and publicized by Alice Hamilton in her classic treatise.[11]

CHILDREN - THE BAROMETER OF ENVIRONMENTAL LEAD

The discovery by Kettering and by Midgely in 1921 of tetraethyl lead as a highly effective antiknock agent in internal combustion engines resulted in a new mode of atmospheric dispersion. The regulatory history of the alkyl leads also provides a cautionary tale. The U.S. Public Health Service, charged with the protection of workers and the general public, recognized the neurotoxic hazard of the organic leads and imposed stringent controls on the transport and labeling of tetraethyl lead. It was decided, however, on the basis of inadequate animal tests, that gasoline exhaust posed no hazard and no restrictions were placed on either the automotive use of tetraethyl lead or on the burning of alkyl leads in other fuels.[12]

Natural Levels of Lead

This new source of environmental lead contamination is indelibly charted on the lead analysis of glacial snow (Fig.2) which document the increased aerosol fallout of lead with increasing use of automobiles and airplanes.[13]

These analyses of serial depositions of silicate dusts in polar snow are one of several methods employed to determine the natural concentration of lead in air, water, and food in premetallurgic times. Another paleontologic technique has been the analysis of the lead:calcium ratio. As defined by Settle and Patterson,[14] the natural cycle of land from rock, water, and soil to plants, to herbivores and then to carnivores involves a biopurification process due to the selective absorption of calcium over lead that is calculated as resulting in a 1000 fold difference.

As shown in Table 1 this would predict that the average lead:calcium ratio of the earth's crust is reduced from $6.4x10^{-5}$ to a lead:calcium ratio of $3x10^{-8}$ in prehistoric humans and other carnivores. Ericson, et al. employing ultra clean techniques, found this predicted value to be similar to the lead:calcium ratio of human bones from premetallurgic Peru.[15] Bone analyses on contemporary British and American adults showed lead:calcium ratios 200 to 600 times higher than those of ancient Peruvians.

A third approach to environmental contamination with lead has been the direct analysis of human bones. Although all such data is subject to considerable error from environmental contamination as well as the variables of analysis, there is an increase in the concentration of lead in human bones (Fig.3) with the thousand fold increase in production about 2500 years b.p. (Fig.1) Human bones obtained from premetallurgical societies in Peru,[15] Egypt, Nubia, and ancient Denmark[16] all have lead concentrations below 3 ug/g. In contrast, the concentrations of lead in English bones from the early days of the Roman empire are elevated 10 fold and increase to 300-400 ug/g, by the time of the Norman Conquest.[17] Bone lead concentrations above 200 ug/g are generally considered unequivocal evidence of chronic lead poisoning. Grandjean's analyses of Danish bones show similar increases in medieval times and peak concentrations in the 18th century.[16] The content of lead in bone increases both with age and with bone density. Barry, in analyses of bones from contemporary English children, 1-5 years old, found vertebral levels of 0.3-6.5 ug/g, increasing to 1-9 ug/g at 11-16 years of age.[18] The values for contemporary adults are also widely divergent; they range from the 0.1-5.4 ug/g reported by Grandjean in Denmark to 7.5-195 ug/g as reported by Schroeder and Tipton in data from U.S. adults dying in the 1950's.[19] Despite analytic errors, there is compelling evidence from multiple sources that the contemporary urban American population absorbs at least 100 times as much lead as that of premetallurgical population and that the body burden is close to the detectable toxic thresholds for lead.

Contemporary Epidemiology

Concern with the sources and level of environmental contamination by lead is far more than an academic exercise. In any individual case immediate inquiry must be made concerning the incidence of pica, mouthing behavior, and the chronic ingestion of a multitude of high lead sources including the following:[20]

HOUSEHOLD SOURCES OF LEAD

ORAL

- Paint, plaster
- Painted toys, cribs, railings
- House dust
- Soil - yard and street
- News print
- Lead water pipes (only if water pH low)
- Teakettles, water heaters, boilers (lead solder)
- Toothpaste
- Canned foods
- Milk - animal and human
- Wine, homemade
- Moonshine (from lead solder in car radiators and home stills)
- Glazed pottery and painted glassware
- Pewter pitchers and utensils
- Herbal medicine
- Cosmetics, oriental

INHALED

- Stoves burning battery cases, wood/nail rubble and recycled oils
- Lead shot reloading

Industrial sources of lead, listed below, can pose a significant risk for children and adults by mass contamination of air and soil, as well as the heavily contaminated dust transported by the parent's clothing and car.

MAJOR INDUSTRIAL SOURCES OF LEAD

- Welding and cutting of lead painted metal
- Lead mining, smelting and refining
- Battery reclamation
- Brass alloy foundries
- Lead pigment production
- PVC plastics (lead salt stabilizers)
- Spray painting
- Soldering, solder grinding
- Auto and ship repair
- Lead crystal making
- Lead casting and type setti
- Urban renewal projects

CHILDREN - THE BAROMETER OF ENVIRONMENTAL LEAD

The U.S. lead industry produces about 1.3 million metric tons per year. Approximately one half, primarily that in batteries and other metallic products, is reclaimed from scrap with some general contamination of air, water, and soil during the recycling process. General environmental contamination with lead is increased by an estimated 600,000 metric tons annually in the form of pigments, gasoline additives, recycled petroleum products, ammunition, and other products not suited to reclamation.

As shown in the diagramatic scheme of Fig. 4, industrial fallout is heaviest in the urban environment, absorption is highest in the child and biologic sensitivity is greatest in the young child.[20] At the time of implementation of the Lead Paint Poison Prevention Act in 1970 it was assumed that the elimination of substandard housing characterized by abnormally high lead in deteriorating surfaces of paint and plaster would eradicate lead poisoning. Data analyses, however, show no identifiable source of lead paint exposure in 32-45% of children with elevated blood levels.[21] Other studies have shown a higher correlation of blood lead in preschool children with age, pica, mouthing behavior and sociocultural factors than with the lead content of household paint.[22] It is apparent that the general urban contamination with lead is the environmental risk factor for children, and the relative contributions of air, dust fall, soil, house dust, water, food and other sources have been the subject of multiple investigations.

Our own inquiry into the sources of environmental contamination began with the 1967 report of the National Air Surveillence Network showing that Omaha had the highest dustfall lead of 22 midwestern cities. This exposure was attributed to the presence of one of the largest lead refineries in the country plus two other industrial emission sources, all in close proximity to target areas of urban poverty.[23-27] (Fig.5) Windrose data showed the prevailing winds to focus the emissions on this north central urban area. Since rational environmental standards require epidemiologic data, a long term study was initiated in Omaha of the correlation of childhood blood lead with the lead content of air, soil, house dust, water, and milk. The majority of our studies involved the age group 6-11 years in order to eliminate the variables of differences in mouthing behavior and pica. Studies were initiated at schools in north central Omaha and later at suburban schools to assess blood and environmental lead in three general areas and populations. C: a commercial area in north central Omaha with an elementary school directly adjacent to a battery plant. M: a mixed commercial residential area also in the north central target area of industrial emissions and poverty. S: suburban southwest Omaha. Almost all children and youth tested in areas C and M were black; those in S were predominantly white.

Air lead samples were collected as 24 hour collections at 6 day intervals in High-Vol samples (0.1-1.0 μ/particle size) at fifteen feet elevation at three sites. Previous studies had shown no significant difference between air lead collected at 4' and 15'. Monthly dustfall was collected in plastic containers at the same sites as the air samples. Soil samples were collected as two inch cores halfway between the building and the lot line for each individual home and each school. Household dust was collected from vacuum cleaner bags.

The range of concentrations of lead from these sources are shown in Table 2 which compares the levels in air, soil, house dust, and water in these areas of Omaha with that of national samples.[28]

CHILDREN - THE BAROMETER OF ENVIRONMENTAL LEAD

An early epidemiologic study investigated the correlation of the blood lead of urban school children 6-11 years at C and M with residence in census tracts characterized by substandard housing. (Fig.6) A topographic plot of the blood lead of school children according to their home residence showed blood lead to be unrelated to housing. (Fig.7) The highest blood leads occurred in children living in the immediate proximity of the small battery plant and those living along the major trafficways.[25,26]

Serial sampling of blood and environmental lead showed significant decreases over time.[27] (Fig.8) In 1971 blood lead of school children living in the immediate vicinity of a battery plant averaged 37 ug/dl, now considered in the toxic range. By 1977, the average blood lead had decreased to 22 ug/dl in urban Omaha and to 14 ug/dl in suburban children.

During the same period of time the geometric mean of air lead decreased as follows at each of the three sites C (commercial-battery plant), M (mixed urban) and S (suburban); although there was an increase in 1977 associated with an increase in total particulates.

AIR LEAD ug/m^3

	C	M	S
1970	1.66	1.44	0.73
1972	0.37	0.32	0.26
1973	0.46	0.32	0.22
1974	0.04	—	0.04
1976	0.13	0.16	—
1977	0.78	0.62	—

Dust fall lead, expressed as $mg/m^2/30d$, were as follows, with the number of samples in parentheses:

	C(9)	M(10)	S(7)
1973	26	11	7
1974	17	6	3
1976	5	1	-
1977	34	8	-

The geometric mean and ranges of soil Pb (μg/g), with the number of samples in parentheses, were as follows:

	GM	Range
Site C (69)	262	53-1615
Site M (56)	339	20-4792
Site S (51)	81	16-341

House dust lead (μg/g) at the three sites showed no significant urban-suburban difference in the geometric mean (number of samples in parentheses):

	GM	Range
Site C (26)	479	76-5571
Site M (14)	300	76-860
Site S (26)	211	18-845

Milk and water study analyses had shown no urban-suburban difference with all milk in Omaha coming from a common milkshed. Similarly, all water samples, collected as 30 day samples from 20 homes, had lead concentrations below 10 μg/l. This low concentration of lead is consistent with the high pH (8.5 to 9.5) and the low plumbosolvency of Omaha tap water.

The major grocery chains in Omaha are equally distributed in the three areas and food analysis was not a part of the study.

The data were then analyzed for the age groups 1-5 years and 6-18 years for urban and suburban children to determine the correlation of blood lead with concentrations in air, soil, and house dust. Significant bivariate correlations are given in Table 3.

Significant correlations of blood lead with air lead were found only in the age group 6-18 years old. This equation predicts an increase in blood lead of 1.4 μg/dl as the air lead increased from 1-2 μg/m^3. This is compatible with experimental studies in adults by Chamberlain, et al[31] and Griffin et al[32], which showed an increase in blood lead of 1.2 to 1.7 μg/dl as air lead increased by 1 μg/m^3. Yankel et al, in a 1977 study of children in the Idaho smelting area, derived a multiple regression equation that predicts an increase of blood lead of 1.1 μg/dl as air lead increases from 1-2 μg/dl at a constant level of soil and dust fall lead.[33]

The bivariate correlation of blood lead with soil and house dust lead predicts an increase in blood lead of 5.2 μg/dl as the soil lead increases from 100 to 500 μg/gm and predicts an increase in blood lead of 5.3 μg/dl as the house dust increases from 100 to 500 μg/g.

A multivariate correlation of blood lead (PbB) with the lead content of air (PbA), soil (PbS), and house dust lead (PbHD) was derived:

$$PbB = Bk\ Pb\ A^{0.03}\ PbS^{0.10}\ PbHD^{0.07}$$

This Omaha equation (Table 4) was applied to four other community studies[33-36] and showed an excellent prediction of blood lead with a relatively narrow range of background lead (Bk) of 4.8 to 8.1 μg/dl.

CHILDREN - THE BAROMETER OF ENVIRONMENTAL LEAD

Charney, Sayre, and Coulter have recently re-examined their long term environmental studies in Rochester, NY.[22] Stepwise correlations establish the lead content of household dust and of the dust on the hands, mouthing behavior and pica as the critical variables in blood lead for children under 6 years of age. No single factor accounted for more than 50% of the variance in blood lead.

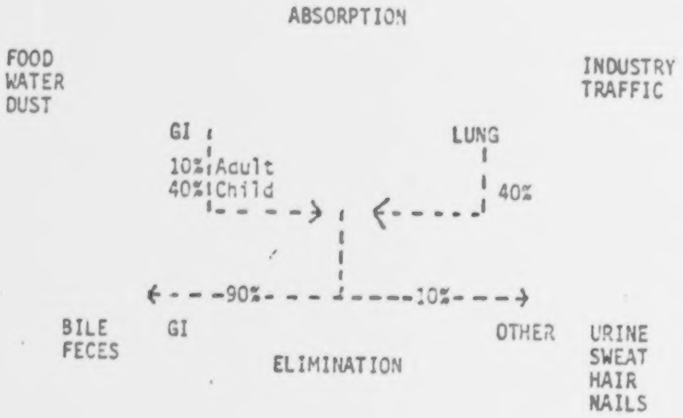
Roels et al, in their long term studies of Belgian children attending schools at 1 and 2.5 km from a lead smelter and in urban and rural areas have shown the high correlation of blood lead with playground dust and air lead.[38] The lead on the children's hands was significantly related to the environmental values. Partial correlations showed that the lead on the hands (PbH) was quantitatively the major contributing factor (r=0.976) to blood lead (PbB). The correlation of blood lead and lead on the hands was:

$$PbB = -4.33 + 11.54 \log PbH$$

This equation predicts an increase in blood lead from 18.8 to 22.2 µg/dl as the lead on the hands increases from 100 to 200 µg/gm. Roels et al concluded, as we did, that although the fallout from the air is the starting point of environmental contamination, the enhanced daily intake of lead by the oral route is the primary identifiable factor predicting the blood lead of school children. As observed by Yankel et al,[33] the cleanliness factor of individual homes also contributes to the variance in the blood lead of children of all ages living in areas of similar environmental contamination, just as the amount of mouthing behavior is a major variable in preschool children.

Absorption and Metabolism of Lead

One of the most important conclusions from these multiple epidemiologic studies of blood lead is that the greater intestinal absorption of lead by children than by adults appears to be a significant factor far beyond early infancy. In both children and adults the absorption of inhaled lead of 0.1-0.1 μ/particle size is 35-40% of the respired load.[31,32,39]


ABSORPTION
FOOD
WATER
DUST
INDUSTRY
TRAFFIC
GI
LUNG
10% Adult
40% Child
40%
90%
10%
BILE
FECES
GI
OTHER
URINE
SWEAT
HAIR
NAILS
ELIMINATION

Adults absorb approximately 10% of ingested lead while infants and young children may absorb as much as 40%. Insufficient data are available to define possible age-related differences in biliary secretion.[40]

Estimates of the exposure and net absorption in children are given in Table 5:[28,39,41]

CHILDREN - THE BAROMETER OF ENVIRONMENTAL LEAD

Mahaffey indicates that the maximal permissable exposure of young children to lead should not be greater than 100-150 μg/day.[40] Ziegler et al, concluded from individual balance studies in children 2-25 months old that an intake of food lead of less than 50 μg/day may achieve a negative lead balance.[42]

Absorbed lead is transported in the blood with about 95% bound to hemoglobin. Plasma lead rarely exceeds 2-3 μg/dl. The binding of a small but variable amount of lead to a low molecular weight protein of the red cell has been described by Raghavan and Gonick.[43] Experiments in human volunteers by Chamberlain showed that the blood lead peaks about one day after injection, ingestion or inhalation.[44] The clearance of lead from the blood seems to follow several rates of decay with one as short as five days and the longest being six months.[45] Soft tissue lead has a turnover time of 30-40 days. Pharmacokinetically, the third and largest compartment is bone lead which accounts for 95% of the total body burden in adults and about 70% in young children.[46] The residence time in bone is at least 30 years. This lifelong skeletal storage is the basis for the EDTA challenge test which will provoke an increased urinary lead many years after termination of exposure. Since blood lead has the most rapid turnover of all of the body compartments, more stable indices, such as erythrocyte protoporphyrin IX (EP), are frequently used to define exposure. The current definitions of excessive lead absorption as given by the CDC[47] are as follows:

Classification Used in Screening Program of the Center for Disease Control

Lead poisoning is defined as existing whenever a child has any one or more of the following:

1. Two successive blood lead levels equal to or greater than 70 µg/dl with or without symptoms.
2. EP level equal to or greater than 250 µg/dl whole blood and a confirmed elevated blood lead level equal to or greater than 50 µg/dl with or without symptoms.
3. EP level greater than 109 µg/dl associated with a confirmed elevated blood lead level ($\leq$30µg/dl) with compatible symptoms.
4. Confirmed blood lead level greater than 49 µg/dl with compatible symptoms and evidence of toxicity (e.g. abnormal EP, calcium disodium EDTA mobilization test, urinary aminolevulinic acid excretion or urinary coproporphyrin excretion).

Lead toxicity: erythrocyte protoporphyrin (EP) equal to or greater than ($\geq$) 50 µg/dl or functional derangements caused by lead.

Undue lead absorption: confirmed blood lead levels of 30-69 µg/dl associated with EP levels of 50-249 µg/dl whole blood.

Elevated blood lead: a confirmed blood lead of 30 µg/dl or greater.

Biotoxicity of Lead

Lead interferes with the synthesis of the heme by enzymatic inhibition of three major enzymes:[48] 1) delta-amino levulinic acid synthetase (ALA-S), the mitochondrial enzyme that catalyses the fusion of glycine and succinyl CoA to form delta-amino levulinic acid (ALA), the urinary excretion of which is a useful index of exposure; 2) ALA-dehydratase, the cytosolic enzyme responsible for the linkage of two molecules of ALA to form the single porphyrin ring of porphobilinogen (PBG). PBG is then incorporated into the successive 4-ring molecules of uroporphyrinogen III, coproporphyrinogen III, protoporphyrinogen IX and protoporphyrin IX; 3) ferrochelatase, a mitochondrional enzyme, and therefore found in liver, marrow and other tissue, but not in the mature red cell. Ferrochelatase activates the incorporation of the ferrous ion into the center of the protophyrin ring with formation of heme. Since hemoglobin synthesis is heme dependent, lead inhibits the formation of hemoglobin as well as the heme dependent proteins of the P450, b5 and other cytochromes of hepatic mitochondria.

Red cell protoporphyrins will be elevated in the peripheral blood at blood lead levels as low as 20 μg/dl and persist for the life span of the red cell, thus reflecting the average exposure over three months.

The usual assay of erythrocyte protoporphyrins (EP) measures protoporphyrin IX, coproporphyrin III, uroporphyrin I and possible others. Assay of free erythrocyte protoporphyrins (FEP), so named under the mistaken impression they were metal free, primarily measures protoporphyrin IX and coproporphyrin III. The hematocytofluorometers used in most lead screening programs measure the ratio of fluorescence to hemoglobin as compared to a standard of zinc protoporphyrin (ZPP). Validity is affected by hemoglobin concentrations and the presence of unoxygenated or methemglobin, bilirubin and other substances. FEP is elevated in iron deficiency anemia, erythrocyte prophyrinurias, febrile chronic infections and other disease and deficiency states.[49]

A summation of the thresholds for specific biologic changes in children is as follows:[28,51]

Blood Lead Level (μg Pb/dl)	Effect
0-10	Py-5' nucleotidase inhibition
10	ALAD inhibition
15-20	Erythrocyte protoporphyrin elevation
40	Increased urinary ALA excretion
40	Anemia
40	Coproporphyrin elevation
50-60	Cognitive (CNS) deficits
50-60	Peripheral neuropathies
50-70	Lead lines in bones
80-100	Encephalopathic symptoms

CHILDREN - THE BAROMETER OF ENVIRONMENTAL LEAD

Our own studies of the subclinical effects of lead include investigations of its inhibitory effect on pyrimidine-5'-nucleotidase (Py-5N)[50-54] in the red cell cytosol. This enzyme has a specific affinity for the pyrimidine nucleotides, predominantly uridine monophosphate and cytidine monophosphate. Phosphorolysis allows the parent nucleosides, uridine and cytidine to diffuse from the cell thus permitting selective conservation of the adenine and guanine phosphates essential to energy metabolism. Congenital deficiency of pyrimidine-5'-nucleotidase is characterized by a hemolytic anemia with basophilic stippling morphologically similar to that of lead poisoning.[55,56] In both cases the stippling is thought to represent aggregation of ribosomes in the presence of altered cationic charge, with stippling reversible on brief incubation of the blood with EDTA. With homozygous deficiency of the enzyme to less than 10% activity there is a massive accumulation of red cell pyrimidine nucleotides in excess of 1 mMol/L. In children with excess exposure to lead there is a direct correlation of red cell pyrimidine-5'-nucleotidase with blood lead. (Fig.9) Pyrimidine nucleotides appear at only 50% inhibition of pyrimidine-5'-nucleotidase in lead poisoning (Fig.10) although the absolute amounts are only a fraction of those found in congenital deficiency.[53] In lead poisoning, therapeutic chelation promptly restores activity of pyrimidine-5'-nucleotidase; this reactivation reflects the temporary decrease in blood lead.[54] (Fig.11) Chelation does not decrease the red cell pyrimidine nucleotides, supporting other experimental evidence that the residue of pyrimidine nucleotides relates more to the level of enzyme activity in the reticulocyte than in the mature red cell. The accumulation of erythrocyte pyrimidines thus reflects the net exposure to lead during the antecedent three months. Familial studies of congenital deficiency of pyrimidine-5'-nucleotidase show an increase of mild mental retardation.[55] Any possible association between this observation and the neurologic effects of lead is, at this point, entirely speculative.

The major question concerning asymptomatic increases in blood lead to 20-40 µg/dl is whether the association with mental retardation is causal or an indirect effect related to sociocultural factors. This is the subject of intense investigation as reported by Needleman,[57,58] analysis as reviewed by Cowan and Leviton[59] and controversy, as noted by Ernhart et al.[60] Clinical lead poisoning is associated with a significant risk of gross neurologic deficit.[61,62] In one of our early studies of children with lead encephalopathy, we found 87% of children to have gross neurologic sequelae.[63] Perlstein and Atalla,[64] in 1966, cited mental retardation in 24% of children with clinical lead poisoning and in 9% of those with blood lead over 60 µg/dl but without toxic symptoms.

Provocative insight into the possible interaction of the effect of lead on heme synthesis with its neurotoxicity is provided by Silbergeld and Lamon[65] who note the chemical similarity of ALA (COOH-C-C-C-C-NH_2) and the neurotransmitter, GABA (COOH-C-C-C-NH_2) and the affinity of ALA for GABA receptors of rat synaptosomes. The possibility that the neurotoxicity is mediated by aberrant heme metabolism would help explain some of its unusual aspects: the lag time in production of neurologic symptoms, the incongruity of early deficits in affect and cognitive function with localization of increased lead in the cerebellum and the better correlation of neurobehavioral function with protoporphyrin levels than blood lead, and might also elucidate the similarity to the neurotoxic prophyrias.

In addition to the effect of lead on red cell and neural tissue as the primary target organs, lead exerts a toxic effect on almost every organ system.

ENVIRONMENTAL LEAD AND CHILDREN: THE OMAHA STUDY

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Pesticides B..
Dallas, Texas

Blood lead (Pb B) was determined in 1232 samples from 831 children in Omaha and correlated with air lead (Pb A) concentrations of 0.02-1.69 μg/m³ from 1971 to 1977. A bivariate equation for ages 6-18 yr based on these data predicts an increase in Pb B of 1.4 μg/dl as Pb A increases from 1 to 2 μg/m³. Pb B increases 7 μg/dl as the mean values for soil and house dust Pb increase from 100 to 750 μg/g. Multiple regression analysis shows that the combined effects of air, soil, and house dust Pb account for 21% of the variance of Pb B, with a high intercorrelation of all 3 variables. Since the variance of repeat sampling in individuals accounted for 38% of the total variance of Pb B, approximately 40% is unexplained and requires measurement of Pb from dietary and other sources.

INTRODUCTION

The Environmental Protection Agency (1978) established the air quality standard for Pb at 1.5 μg/m³:

> This level was derived from the Agency's judgment that the maximum safe blood lead level (geometric mean) for a population of young children was 15 μg Pb/dl and, of this amount, 12 μg Pb/dl should be attributed to nonair sources. The difference of 3.0 μg Pb/dl was estimated to be the allowable safe contribution to mean population blood lead from lead in the air. With epidemiological data indicating a general relationship of 1:2

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between air lead ($\mu g\ Pb/m^3$) and blood lead ($\mu g\ Pb/dl$), EPA determined that the level for the proposed standard should be 1.5 $\mu g/m^3$.

This standard was based on the assumption of a crude average of background Pb, as data were insufficient "to predict the relationship between air lead levels, dustfall rates and resulting soil accumulation." Only four of the reported childhood studies—Roberts et al. (1974) in Toronto, Johnson et al. (1976) in California, Yankel et al. (1977) and Ashley (1976) in Idaho, and the El Paso study [*Morbidity and Mortality Monthly Report* (*MMMR*), 1978]—included data on soil and dust as well as air as related to blood Pb.

We have been monitoring air Pb in Omaha at three sites since 1970, simultaneously sampling house dust, soil, and dustfall Pb, for correlation with 1232 samples of blood lead (Pb B) from 831 preschool, school-age, and high school children (McIntire and Angle, 1972; Angle et al., 1974, 1975a–1975c; Angle and McIntire, 1975). This is a report of the correlation of childhood Pb B with air, soil, and house dust Pb as found in the 7-yr Omaha study.

METHODS

Subjects

Children participating in the study were all volunteers recruited with the cooperation of their schools and parents. Much of the support was related to public enthusiasm for the Clean Air Act of 1970. Throughout the study the children are categorized by school level and age: preschool, ages 1–5 yr, $n = 199$ (244 samples); grade school, junior and senior high school, ages 6–18; $n = 632$ (988 samples). Preschool children were sampled in 1974 only, whereas sampling of older children was distributed over 6 yr. The children live in three general areas in Omaha: urban-commercial (C), $n = 132$, in the vicinity of a small battery plant; urban-mixed (M), $n = 589$, a residential area contiguous with downtown Omaha; and suburban (S), $n = 110$, as shown in Fig. 1. There is potential bias in the selection of subjects since the children are all volunteers and subjects are not equally distributed among the three sites. Virtually all of the urban children (C and M) are black; all of the suburban children (S) are white. Our earlier research interests resulted in an initial selection of urban black children with a deficiency of red cell glucose-6-phosphate dehydrogenase. Data on these children are retained since this enzymatic defect has no effect on Pb B, although an increase in red cell Pb coexists with decreases in hemoglobin (Angle and McIntire, 1975). In the suburban high school population there is an excess of males; elsewhere there is an equal sex distribution of the subjects.

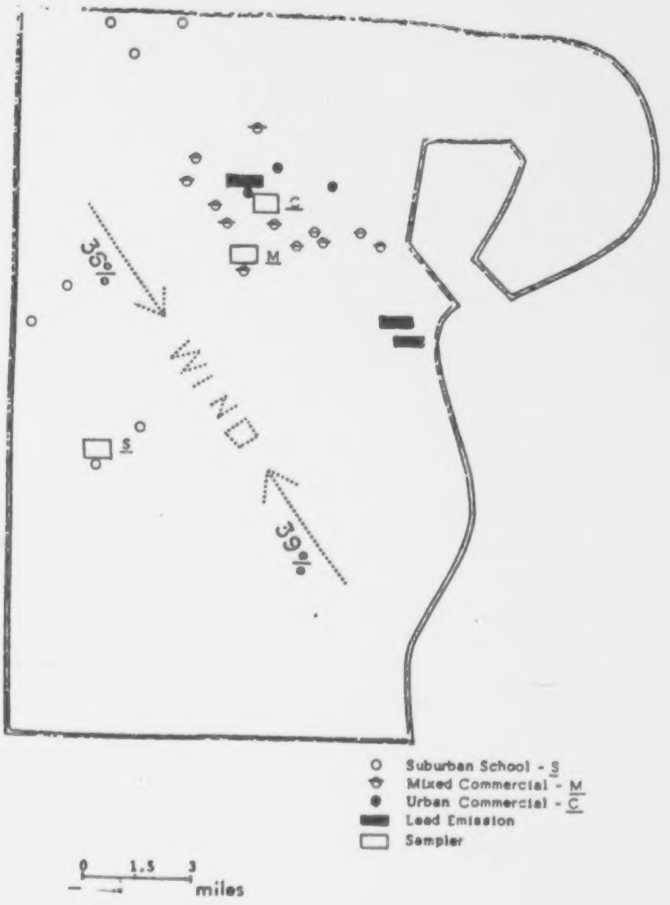
35%
WIND
39%
C
M
S
○ Suburban School - S
Mixed Commercial - M
● Urban Commercial - C
Lead Emission
☐ Sampler
0 1.5 3
miles

FIGURE 1. Map of Omaha showing the commercial (C), mixed commercial-residential (M), and suburban (S) sampling sites and schools as related to three Pb emission sources.

Blood Lead

In 1971, all samples were venous blood (collected in Pb-free heparinized B-D Vacutainers), analyzed by the macro method of Farrelly and Pybus (1969). In 1972 and 1973, all subjects had both a macro Pb B determination and a capillary Pb B (acid-washed capillary tube transferred to a B-D Unipette) determination by the Delves (1970) micro cup method to establish the validity of the micro assay. Assays done at this time showed that the micro leads had a smaller standard error (3.2 versus 3.8 µg/dl) but were consistently higher by 4.0 µg/dl. The macro Pb B values of 1971 were corrected to the higher micro values. All assays from 1972 to 1977 were done by the Delves micro cup method, using a Perkin-Elmer model 303 atomic absorption spectrophotometer. All assays from 1971 to 1977 were done in replicate, employing the method of addition for each individual sample.

Air Lead

Air lead (Pb A) samples were obtained as 24-h collections at 6-d intervals in Hi-Vol samplers (particle size 0.1–10 μm) at 15 ft elevation at schools in each of the 3 geographic areas. Previous studies showed no significant difference in the Pb A at 3 and 15 ft (Angle et al., 1974). The sampler at site C was approximately 500 yd from the emission source; all samplers were more than 15 yd from a roadway. In 1970 and 1971 the filters were assayed for Pb A by R. E. Enrione (Metals and Advanced Analysis Section, EPA, Cincinnati, Ohio). In 1972 and 1973 assays were done by E. R. Williams (South Carolina Department of Public Health under contract to the EPA). In 1974–1977, Pb A assays were done by the Omaha-Douglas County Health Department by atomic absorption spectrometry (AAS), using the method of Purdue et al. (1973). Concordance of Pb A values for 1973 was used to establish the reliability of the assays done by the Omaha laboratory. The values in 1971 were reported by Enrione as the composite of all filters for that month. Subsequent monthly means were derived from the individual values of filters every sixth day.

Dustfall Lead

Dustfall Pb was collected by the standard technique of total accumulation in a plastic container at a height of 4 ft at each of the 3 air collection sites and reported as milligrams of Pb deposited per square meter per 30-d collection. Dustfall, soil, and house dust were measured by AAS in the same laboratories as Pb A.

Soil Lead

All samples of soil Pb were collected as a 2-in core obtained halfway between the building and the lot line on all 4 sides and reported as the arithmetic mean. In 1972, soil and household Pb samples were collected from 37 individual houses for correlation with the Pb B of individual children. An additional 148 samples were taken at each child's school or preschool, with a total of 20 sites sampled 1 to 5 times.

Household Dust

Household dust was obtained at the same sites as soil. At most sites it was obtained by emptying the vacuum cleaner bag. Where there was no vacuum cleaner a composite was obtained by sweeping the floor and windowsills.

Milk and Water

As reported by Angle et al. (1974), there was a low level of Pb (<40 µg/l) in the bottled milk supply in Omaha, all of which comes from a common milk shed. Water samples, 30-d collections at 20 individual homes, showed values consistently below 10 µg/l. This low concentration of Pb is consistent with the high pH (8.5-9.5) of Omaha tap water. The major grocery chains in Omaha are equally distributed in the three areas and food analysis was not part of the study.

Statistical Analysis

Arithmetic and geometric group means were determined for blood, air, dustfall, soil, and house dust. Variance attributed to sampling error was determined by the method of Lucas (1977) and expressed as the percentage of total variation that derives from the deviation of repeat values from the average for the individual subject.

Correlations based on group means were considered unsatisfactory because of the differences between the groups sampled each year. All 1232 samples were used to derive the variance of Pb B. Of the total 1232 samples, 1074 had adequate data on air, soil, and dust. Environmental values were assigned to these 1074 samples of Pb B from 831 children as follows: (1) Pb A was the monthly geometric mean of the closest sampling within the prior 3 mo; (2) dustfall Pb was the monthly geometric mean of the closest sampling within the prior 6 mo; and (3) soil and house dust Pb was the geometric group mean according to school within 2 yr of the blood sampling. For the 37 subjects with individual values for household and soil Pb, the individual means were retained. There was a small number of house dust and soil samples for 1975-1977, but little difference was observed between these values and the means for the preceding years. All environmental values are expressed as geometric means. Employing these individual environmental values, we calculated linear and log-log (base 10) intercorrelations of environmental Pb and simple and multiple regression equations for the correlation of Pb B with environmental Pb for the total and for each subgroup according to age and location. The log-log regressions were more consistent and are employed throughout. All statistical analyses were done by the methods in Nie et al. (1975).

RESULTS

Blood Lead

Pb B as measured in 1232 samples from 831 subjects was not normally distributed (mean ± SD, 22.7 ± 8.3 µg/dl; skew, 1.59; kurtosis, 3.59) but was close to log-normal (geometric mean, 21.47 µg/dl; standard geometric deviation, 1.395; skew, 0.36; kurtosis, 3.0). In a normal distribution, skew = 0 and kurtosis = 3.0.

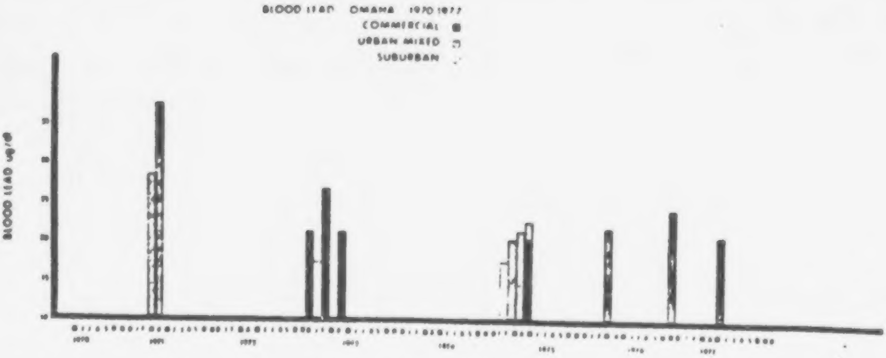

FIGURE 2. Pb B (arithmetic means) of 1232 samples from 831 children 1-18 yr old, 1971-1977.

Group distributions of Pb B showed the geometric mean and standard geometric deviation (GM ± SGD) of 24.8 ± 1.4 at site C to be significantly ($p < 0.001$) higher than the value of 22.0 ± 1.4 at site M, which was higher ($p < 0.001$) than the value of 18.0 ± 1.3 at site S. As shown in Fig. 2, Pb B decreased from 1971 to 1975 and there was no significant change from 1975 to 1977.

Analysis of the variance of Pb B (Table 1) showed that sampling variance, or the difference between repeat samples from one individual, accounted for 38% of total variance. Since all samples were done in replicate by the method of addition, no additional estimate is made of analytic variance.

The higher Pb B from May through October suggested seasonal changes as one component of sampling variance, but in the absence of a 12-mo sampling of a comparable age group at one site, seasonal variance could not be validated.

TABLE 1. Variance of Pb B

Age (yr)	Individuals	Samples	$\bar{X}$ ± SD (µg/dl)	Total variance[a]	Intra-individual variance[b]	Between-individual variance[c]
1-5	199	244	23.9 ± 9.2	84.6	37.4 (44%)	56%
6-18	632	988	22.3 ± 7.7	60.1	27.7 (46%)	54%
Total	831	1232	21.3 ± 8.1	65.6	25.2 (38%)	62%

[a] Total variance is the square of the standard deviation.

[b] Intra-individual variance = $\Sigma\,(X_i - \bar{X}_i)^2/[(r-1)/n]$, where X_i is a single sample (replicate average) from an individual, $\bar{X}_i$ is the average of all samples from the individual, r is the number of repeat samples for the individual, and n is the number of individuals.

[c] Between-individual variance is total variance (100%) minus individual variance. Calculations are derived from Lucas (1977).

Air Lead

Maximum monthly Pb A was 4.8 µg/m³ at C, 2.3 µg/m³ at M, and 1.6 µg/m³ at S. Annual geometric mean Pb A (µg/m³) at each site was:

	C	M	S
1970	1.66	1.44	0.73
1972	0.37	0.32	0.26
1973	0.46	0.32	0.22
1974	0.04	–	0.04
1976	0.13	0.16	–
1977	0.78	0.62	–

The seasonal pattern, graphed as the monthly arithmetic mean in Fig. 3, showed peak values in January through March, consistent with seasonal climatic inversions. Since simultaneous blood samples are not available for all levels of Pb A, the range of the monthly geometric mean Pb A assigned to the 1074 blood samples was 0.02–1.69 µg/m³.

Dustfall Lead

The number of samples (given below in parentheses) at each site was limited. The following geometric means of monthly dustfall (mg/m² per 30 d) have the same rank order as Pb A:

	C (9)	M (10)	S (7)
1973	26	11	7
1974	17	6	3
1976	5	1	–
1977	34	8	–

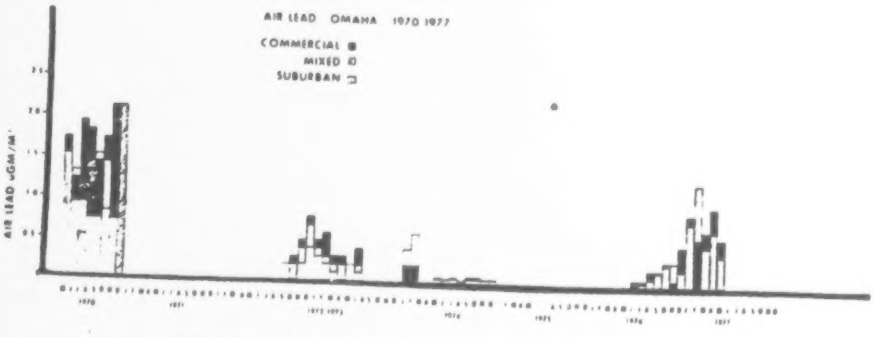

FIGURE 3. Pb A (monthly arithmetic means) at three sites.

Soil Lead

Geometric means and ranges of soil Pb (μg/g), with the number of samples in parentheses, were as follows:

	GM	Range
Site C (69)	262	53-1615
Site M (56)	339	20-4792
Site S (51)	81	16-341

The geometric mean soil Pb assigned to 1074 blood samples was 227 μg/g with a ninety-fifth percentile of 843 μg/g.

House Dust Lead

House dust Pb (μg/g) at the three sites showed no significant urban-suburban difference in the geometric mean (number of samples in parentheses):

	GM	Range
Site C (26)	479	76-5571
Site M (14)	300	76-860
Site S (26)	211	18-845

The geometric mean of house dust Pb assigned to the 1074 samples was 337 μg/g with a ninety-fifth percentile of 894 μg/g.

Environmental Correlation

Simple Pearson correlations showed a distinct difference in the log-log correlation of Pb B with the Pb A and dustfall lead (Pb DF) between preschool and school-age children, although correlations with soil (Pb S) and house dust (Pb HD) were similar:

	Pb A	Pb DF	Pb S	Pb HD
Pb B, 1-5 yr	−0.44	−0.30	0.27	0.29
Pb B, 6-18 yr	0.31	0.04	0.40	0.29

There was no significant difference in correlation according to year of collection, race, or sex. Differences between urban and suburban children are discussed in connection with the bivariate correlations.

Pearson correlations (log-log) showed a significant intercorrelation of all the environmental variables:

	Pb DF	Pb S	Pb HD
Pb A	0.20	0.37	0.17
Pb DF		0.35	0.57
Pb S			0.27

Because of the small number of dustfall samples and their negligible contribution to the correlations with Pb B, dustfall was excluded from subsequent analyses.

Bivariate Correlations

Bivariate correlations of Pb B with air, soil, and house dust are given in Table 2. The correlation of Pb B with Pb A was positive and significant only for ages 6-18 and is simplified as $\text{Pb B} = 23.2\ \text{Pb A}^{0.08}$ ($n = 832$, $r = 0.31$; $p < 0.001$). The log-log regression given in Table 2 predicts an increase in Pb B of 1.4 μg/dl as Pb A increases from 1 to 2 $\mu g/m^3$. The bivariate slopes for ages 1-5 and for all suburban children were negative, resulting in a flat slope for the entire group, $\text{Pb B} = 22.3\ \text{Pb A}^{0.03}$ ($n = 1074$, $r = 0.10$; $p < 0.01$), as shown in Fig. 4. The slope of the bivariate regression was steeper for the children 6-12 yr old living in the vicinity of the battery plant than for the total urban and urban-suburban groups of children 6-18 yr old.

Bivariate correlations of Pb B with Pb S were similar and significant for both age groups but not for all subgroups. The correlation of blood with soil, simplified as $\text{Pb B} = 9.5\ \text{Pb S}^{0.15}$ ($n = 1074$, $r = 0.38$; $p < 0.001$), is superimposed on the scattergram of Fig. 5. The equation predicts the following Pb B values as soil Pb increases from 100 to 750 μg/g: 19 and 26 μg/dl, respectively. The regression correlation of blood with soil (Table 2) suggests a steeper slope for children 1-5 yr old than for school-age children, but the larger standard error of the coefficient indicates an exponential range of 0.15-0.25.

The correlation of Pb B with house dust Pb, $\text{Pb B} = 8.2\ \text{Pb HD}^{0.16}$ ($n = 1074$, $r = 0.29$; $p < 0.001$), derived from the scattergram (Fig. 6) predicts the following geometric means: 100 and 750 μg/g Pb HD, and 17 and 24 μg/dl Pb B, respectively. As listed in Table 2, the slope of the regression of Pb B as predicted by house dust Pb is steeper for the preschool children, but again there is a large standard error of the correlation coefficient.

Multiple Regression Analysis

A multiple regression analysis of Pb B as the dependent variable predicted by air (Pb A), soil (Pb S), and house dust (Pb HD) was

TABLE 2. Correlation of Pb B with Environmental Pb

Age (yr)	Samples	Equation	SEE[a]	SE coeff[b]	r[c]
		Pb A			
1-18	1074	log Pb B = 1.3475 + 0.0269 log Pb A	0.1449	0.0081	0.10**
1-5	242	log Pb B = 1.2461 − 0.1331 log Pb A	0.1422	0.0172	−0.44
Urban	204	1.2901 − 0.1150	0.1293	0.0167	−0.43
Suburban	38	1.1316 − 0.0451	0.0837	0.0321	−0.23
6-18	832	log Pb B = 1.3660 + 0.0834 log Pb A	0.1346	0.0089	0.31**
Urban-all	620	1.3823 + 0.0899	0.1363	0.0102	0.33**
Battery plant	153	1.4522 + 0.3196	0.1239	0.0372	0.57**
Suburban	212	1.2591 − 0.0171	0.1132	0.0189	−0.06
		Pb S			
1-18	1074	log Pb B = 0.9766 + 0.1515 log Pb S	0.1348	0.0113	0.38**
1-5	242	log Pb B = 0.8936 + 0.2033 log Pb S	0.1532	0.0474	0.27*
Urban	204	1.2895 + 0.0417	0.1430	0.0491	0.06
Suburban	38	0.5288 + 0.3074	0.0843	0.2559	0.20
6-18	832	log Pb B = 0.9563 + 0.1560 log Pb S	0.1273	0.0112	0.44**
Urban	620	0.9203 + 0.1695	0.1313	0.0147	0.42**
Suburban	212	1.3160 + 0.0231	0.1134	0.0802	0.02
		Pb HD			
1-18	1074	log Pb B = 0.9145 + 0.1655 log Pb HD	0.1393	0.0165	0.29**
1-5	242	log Pb B = 0.7494 + 0.2170 log Pb HD	0.1520	0.0460	0.29**
Urban	204	0.8320 + 0.1979	0.1395	0.0614	0.22*
Suburban	38	1.0555 + 0.0384	0.0848	0.0382	0.17
6-18	832	log Pb B = 0.8557 + 0.1921 log Pb HD	0.1347	0.0207	0.31**
Urban	620	B = 0.7950 + 0.2258	0.1334	0.0216	0.39**
Suburban	212	1.2327 + 0.0151	0.1134	0.0563	0.02

[a] Standard error of the estimate.

[b] Standard error of the coefficient.

[c] Significant and positive at (*) $p < 0.01$ and (**) $p < 0.001$.

employed to investigate the total variance attributed to all three sources:

$$\log \text{Pb B} = a + b \log \text{Pb A} + c \log \text{Pb S} + d \log \text{Pb HD}$$

In the preschool children, sampled only in 1974, the multiple regression showed significant positive correlations only with house dust and soil, which accounted ($r^2 \times 100$) for a total of 11% of the variance of Pb B. For ages 6-18 yr there was a significant positive correlation of Pb B with air, soil, and house dust Pb as entered in this order: Pb B = 8.1 Pb $\text{A}^{0.03}$ Pb $\text{S}^{0.10}$ Pb $\text{HD}^{0.07}$ (standard error of estimate, 1.3; $r = 0.46$; $p < 0.001$). The cumulative increases in $r^2 \times 100$ showed air, soil, and house dust to account for 21% of the variance of Pb B. Because of the high

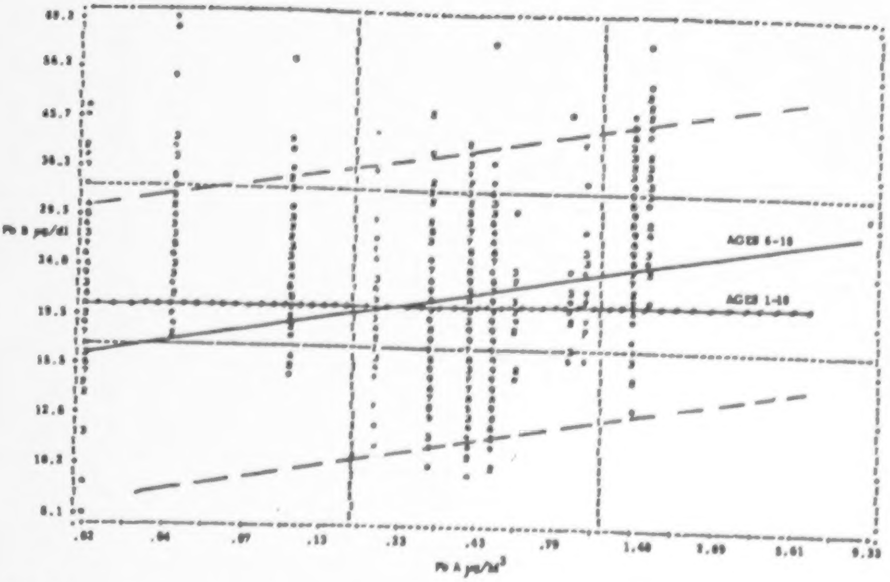

FIGURE 4. Scattergram of the bivariate correlation of Pb A with Pb B in 1074 samples from 831 urban and suburban children 1-18 yr old: Pb B = 22.3 Pb $A^{0.03}$ (dotted line). The correlation for 832 samples from 632 children aged 6-18 is Pb B = 23.2 Pb $A^{0.06}$ ($r = 0.31$; $p < 0.001$) and is drawn as the solid line; dashed lines represent twice the standard error of the estimate. The numbers refer to the number of data points falling at each particular set of coordinates.

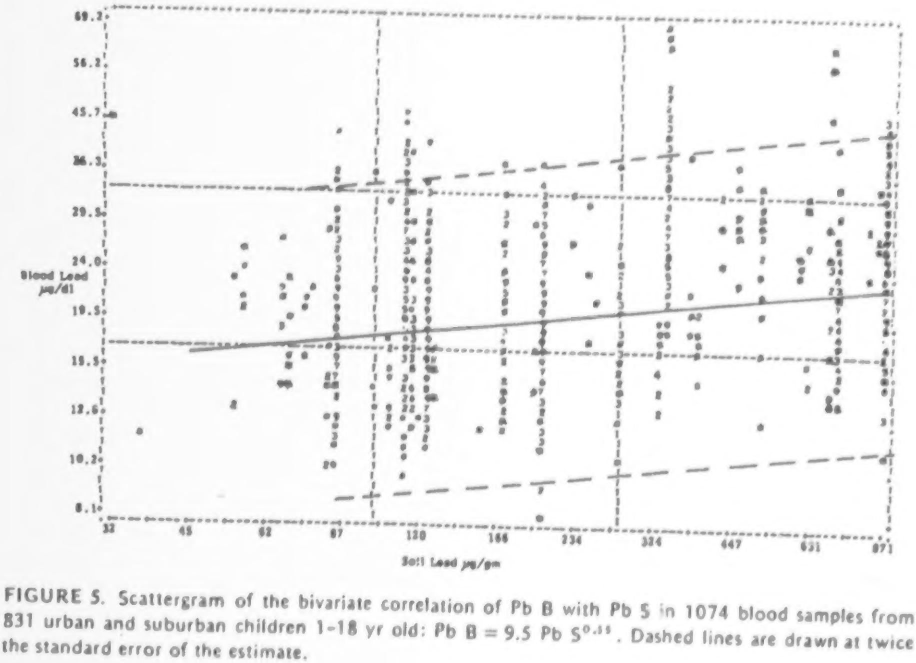

FIGURE 5. Scattergram of the bivariate correlation of Pb B with Pb S in 1074 blood samples from 831 urban and suburban children 1-18 yr old: Pb B = 9.5 Pb $S^{0.15}$. Dashed lines are drawn at twice the standard error of the estimate.

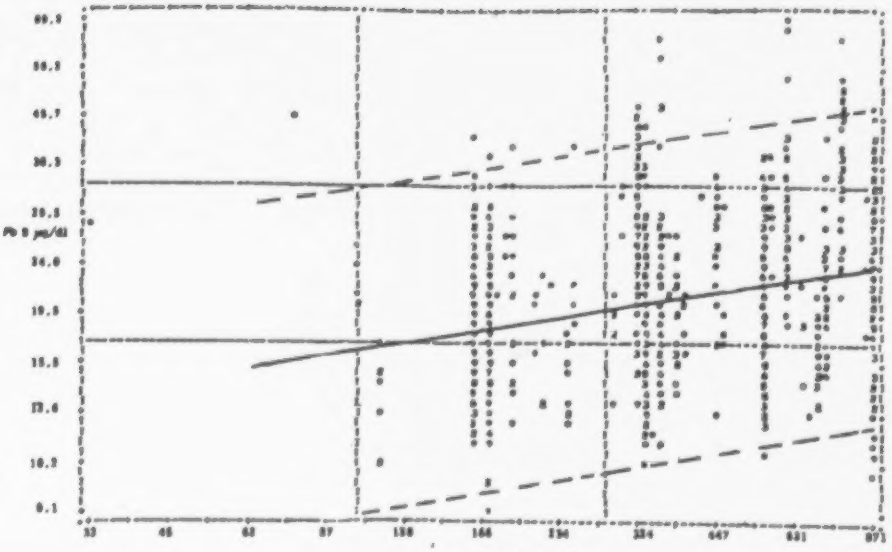

FIGURE 6. Scattergram showing the correlation of 1074 samples of Pb B with Pb HD in 831 children 1-18 yr old: Pb B = 8.2 Pb $HD^{0.16}$. Dashed lines are drawn at twice the standard error of the estimate.

multicollinearity of the independent variables, it was not statistically valid to attribute a discrete percentage to each source.

DISCUSSION

The multiple variables in childhood Pb B and the difficulty of arriving at environmental criteria from community studies are well illustrated by the Omaha study of urban and suburban children in 1971 to 1977. The variables include the nonnormal distribution of Pb B, sampling error, seasonal change, the intrinsic error in any attempt to correlate individual Pb B with environmental samples from communal and stationary sites, and the unmeasured contribution of dietary Pb.

Sampling Variance

As noted in the analysis of variance (Table 1), 38% of the variability in Pb B could be attributed to differences between repeat samples from an individual. The between-individual variance of 62% compares favorably with the 12-61% residual variance that Lucas (1977) found on analysis of the studies of Azar et al. (1975), Tepper and Levin (1975), McLaughlin et al. (1976), and Johnson et al. (1976). The contribution of intrinsic seasonal variation is suggested by the higher Pb B levels of summer, which do not coincide with our seasonal peaks of Pb A, January through March. Air studies in Denver by Edwards and Wheat (1978) showed a similar pattern.

The absence of any significant correlation of Pb B and Pb A in the preschool children was probably caused by the limited duration of sampling, November 1973-July 1974. During this 8-mo sampling period, Pb A was highest in January and February, but there was the usual seasonal increase in Pb B in July. Despite the negative correlation of Pb B and Pb A in this age group, the high intercorrelation of air, soil, and house dust would make it inappropriate to exclude air as a contributing factor to the Pb B of preschool children.

Prediction of Blood Lead

Although neither the bivariate or the multiple regression equations are completely satisfactory from a statistical standpoint, both yield slopes that are in accord with results of other studies. Although the bivariate correlation for ages 6-18, Pb B = 23.2 Pb $A^{0.08}$, is obviously influenced by the simultaneous increase in nonrespiratory sources, the predicted increase in Pb B of 1.4 μg/dl as Pb A increases from 1 to 2 $\mu g/m^3$ is compatible with the general ratio of 1:1 to 2:1 (EPA, 1978). Experimental studies in adults (e.g., Chamberlain et al., 1975; Griffin et al., 1975) under a variety of controlled conditions show an increase in Pb B of 1.2-1.7 μg/dl as Pb A increases by 1 $\mu g/m^3$. The epidemiologic study of adults by Azar et al. (1975) yielded a correlation of Pb B = 16.8 Pb $A^{0.153}$, which predicts an increase in Pb B of 1.8 μg/dl as Pb A increases from 1 to 2 $\mu g/m^3$. Yankel et al. (1977), in their study of children in the Idaho smelting area, derived a multiple regression equation that predicts an increase of Pb B of 1.1 μg/dl as Pb A increases from 1 to 2 μg/dl at a constant level of soil and dust, but analysis (EPA, 1978) of the average response shows an increase of 1.9 μg/dl.

Although the high intercorrelation of the three environmental variables in the Omaha data restricts the delineation of the relative contribution of each variable, the predictive validity of the multiple regression model was tested on crude data from the four community studies of children that included measurements of soil or dust Pb: Toronto (Roberts et al., 1974); Los Angeles (Johnson et al., 1976); Idaho (Ashley, 1976; Yankel et al., 1977); and El Paso (*MMMR*, 1978). As shown in Table 3, the multiple regression Pb B = Bk Pb $A^{0.03}$ Pb $S^{0.03}$ Pb $HD^{0.07}$ (where Bk = background Pb, derived by substituting the blood, air, and dust values at the lower levels for the community) predicts the Pb B within 2 μg/dl in all four communities. This is not submitted as evidence for the universality of the equation but as a demonstration of the interrelationship of inhaled and ingested Pb.

CONCLUSIONS

The Omaha study shows the need to measure the contribution of the dust and soil fallout of airborne Pb as well as dietary and unidentified

TABLE 3. Omaha Equation and Four Community Studies[a]

Study	Pb A (μg/m³)	Pb S (μg/g)	Pb HD (μg/g)	Pb B (μg/dl)	Estimated Bk (μg/dl)	Predicted Pb B (μg/dl)
Idaho	1.0	500	3000	26.7	8.1	26.4
	2.0	1000	3000	28.1		28.9
Los Angeles						
Lancaster	0.64	66	(66)	9.6	4.8	9.7
Los Angeles	6.3	1000	(1000)	14.6		16.4
Toronto						
Control	0.82	99	713	17	6.9	17.2
Smelter	3.01	1715	1550	27		25.1
El Paso						
1977	1.3	427	1479	20.1	6.6	20.3
1972	4.4	1791	22191	31.2		29.4

[a] The regression equation $Pb\ B = Bk\ Pb\ A^{0.03}\ Pb\ S^{0.10}\ Pb\ HD^{0.07}$ was applied to the means of Pb A, Pb S, Pb HD, and Pb B of four epidemiologic studies of children. Idaho data are taken from the predicted equation of Yankel et al. (1977) with the substitution of the average household dust as cited by Ashley (1976, p. 107). Toronto data are from Roberts et al. (1974). The Pb HD cited for El Paso (*MMMR*, 1978) is actually for exterior dust. Los Angeles data (Johnson, 1974) equate soil with house dust. Background Pb B (Bk) was estimated by substituting community data at the lower level of Pb A in the Omaha equation. This Bk was then used to predict Pb B at the higher Pb A. Estimated Bk in Idaho was identical to that in Omaha. The Omaha regression predicts Pb B within 2 μg/dl in all 4 studies.

sources in arriving at environmental standards. Intestinal absorption of Pb may be as high as 42% in children (Ziegler et al., 1978) and, in the absence of pica or unusual sources, dietary Pb is the principal component of the body burden. As reviewed by Mahaffey (1977), dietary intake of Pb by infants averages 75–120 μg/d but may be as high as 716 μg/d.

The determination of the blood-environmental Pb response in community studies is therefore not a matter of a simple correlation, particularly over long periods of surveillance. Community-wide changes in Pb B are multifactorial; air, soil, water, housing, and socioeconomic shifts have an additive or even a synergistic effect. The Lead Paint Poison Prevention Act has a great potential for reducing and ultimately eradicating Pb contamination in housing. The Paint Act and the introduction of unleaded gasoline in 1973 have attacked major components of environmental Pb contamination and may account for the rapid changes in childhood Pb B in large cities (Billick and Gray, 1978). If control of Pb A continues, the existing Pb in the environment will most likely continue to be more important than Pb A in contributing to Pb B levels.

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As reviewed by Eisenger,[45] lead forms complexes with the phosphate groups of nucleic acids, binds reversibly with the sulfhydryl groups of enzymes and other proteins and forms large sized molecular aggregates with protein, such as those readily visible in the renal tubules. Many of the biologic changes are more evident in adults with chronic exposure than in children and are summarized as follows:[28]

Thresholds of Blood Lead Levels for Biochemical and Clinical Effects on Adults

Blood Lead (µg/dl)	Effect
0-10	Inhibition of Pyrimidine-5'-nucleotidase
10	ALAD inhibition
15	Elevation of Erythrocyte protoporphyrin
20-25	Chromosomal abnormalities
30	Toxicity to fetus
30-40	Reduced fertility, altered spermatogenesis
40-80	Anemia
40-50	Increased red cell cytidine triphosphate
40-60	Psychological, sensory, and behavioral changes
40-60	Decreased motor conduction velocity
50-60	Impaired kidney function (acute changes tubular morphology, decreased secretion uric acid)
100-120	Encephalopathic symptoms

Legislation for Environmental Lead

Although more is known about lead than almost any other environmental toxin, there are obvious deficiencies in the available scientific information. As usual in such cases, regulations represent a compromise between maximum protection extrapolated from incomplete scientific data and the political and economic realities. As an example, no effort has been made to regulate the lead content of processed food, although voluntary effort has significantly reduced the lead content of infant foods.[40,41] A summation of current federal regulatory actions governing human exposure to lead is as follows:[28]

Federal Regulation of Environmental Lead

Substance	Source/date	Regulation
Paint	CDC - 1970	Lead Paint Poison Prevention Program for detection, classification and treatment of children 42 CFR 91.
	HUD - 1976	Lead based paint prohibited in housing in which sale, construction or rehabilitation is federally assisted 24.CFR 35.5,35.14,35.24,35.56,35.62
	CPSC - 1978	Lead in paint and surface coatings, furniture, and all toys or other articles for children, maximum .06% (600 ug/g) 16 CFR 1303.4
Utensils	FDA - 1971	Leachable lead, maximum Ceramics, silverplate hollowware, 7 ug/ml Silver, pewter, enamel flatware, small hollowware, 5 ug/ml Large hollowware, 2.5 ug/ml Silver plated baby cups, 0.5 ug/ml Guidelines 7417.00, 7417.01
Air	EPA - 1973	Reduction of lead in gasoline to 0.5 g/gal by 197 40 CFR 80.20 Other criteria of Clean Air Act simultaneously reduced utilization since lead inactivates catalytic convertors
	EPA - 1978	National air quality standard for lead 1.5 ug/m^3, maximum arithmetic mean per quarter 40 CFR 50.12
	OSHA - 1978	Workplace air 50 ug/m^3 maximum, 8 hour average 29 CFR 1910.1025
Fresh Fruits and Vegetables	EPA - 1971	Lead arsensate residue, maximum, 1 ug/g 40 CFR 180.194
Water	EPA - Pending	Drinking water, maximum contaminant lead, 50 ug/l 40 CFR 141.11

The most impressive aspect of the regulatory activities over the past decade has been the associated decline in childhood blood lead. Our own studies in Omaha showed significant decreases in mean blood from 1971-77 that correlated with environmental assays, with quality control considered adequate to rule out decreases in blood lead over time due to variances in methodology. Schneider noted major decreases in the number of symptomatic children disclosed by community lead screening.[66] Even though this data has not been critically analyzed as independent of demographic variables, the current case finding of 4-5% is one-third to one-fifth of earlier surveys. Billick, et al. in New York City noted a decline in the geometric mean blood lead of black children 25-36 months old from 29 µg/dl in 1970 to 21 µg/dl in 1976[67] and HUD data shows a similar trend in Louisville, Kentucky.[28] As the mean blood lead declines so does the risk for symptomatic lead poisoning and its neurologic and other health related sequelae.

The evidence that the regulation of environmental lead correlates with the mean blood lead in children throughout the country establishes the need to continue to monitor blood lead as a barometer of enviromental pollution. The proposed revisions of the Clean Air Act have the potential of increasing ambient lead. The documentation of the health related benefits of decreased environmental exposure to lead provides powerful support for the need to secure the gains of the past decade. Pediatricians and all others concerned with the health of children should urge that this protection be maintained.

CHILDREN - THE BAROMETER OF ENVIRONMENTAL LEAD

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TABLE 1. - PREDICTED AND MEASURED RATIO OF LEAD: CALCIUM IN HUMAN BONES

	Pb/Ca
Earth's crust	6.4×10^{-5}
Predicted, human bones	3.0×10^{-8}
Peru, 1500 b.p.	5.7×10^{-8}
American, present	2.9×10^{-6}

TABLE 2. - REPRESENTATIVE CONCENTRATIONS OF ENVIRONMENTAL LEAD

Air	
U.S., range, urban [28]	0.5 - 2.0 μg/m^3
Omaha, median, 1980 [29]	0.94 μg/m^3
Omaha, median, refinery area 1980 [29]	8.13 μg/m^3
Omaha, median, refinery area 1981 [29]	2.2 μg/m^3
Dustfall	
Omaha, urban 1973-1977 [27]	1 - 26 mg/m^2/30 d
Los Angeles, central [28]	3.2 - 650 mg/m^2/30 d
Los Angeles, suburbs [28]	1.4 - 10 mg/m^2/30 d
Street Dust	
77 midwestern cities [28]	206 - 20,000 μg/g
Soil	
U.S., range [28]	$<$100 - $>$10,000 μg/g
Omaha, urban 1973-1977 [27]	16 - 4792 μg/g
House Dust	
U.S., range [28]	279 - 11,000 μg/g
Omaha, 1973 - 1977 [27]	18 - 5571 μg/g
Water	
95% of 60 U.S. cities [28]	$<$1 - 20 μg/L
Bennington, VT 1977 [30]	50 - 70 μg/L
Food	
EPA survey 1977 [28]	0.1 - 0.5 μg/g

TABLE 3. - CORRELATIONS OF BLOOD LEAD (Pb B) WITH LEAD IN THE AIR (Pb A), SOIL (Pb S) AND HOUSE DUST (Pb HD) IN OMAHA CHILDREN

Age, years	Samples		Standard Error of Estimate	r
		Air Lead		
6-18	832	log Pb B = 1.3660 + 0.0834 log PbA	0.1346	0.31^{x}
6-18 battery plant area	153	log Pb B = 1.4522 + 0.3196 log Pb A	0.1239	0.57^{x}
		Soil Lead		
1-18	1074	log PbB = 0.9766 + 0.1515 log Pb S	0.1348	0.38^{x}
		House Dust Lead		
1-18	1074	log Pb B = 0.9145 + 0.1655 log Pb HD	0.1393	0.29^{x}

^{x} $p < .001$

TABLE 4. THE OMAHA EQUATION AND FOUR COMMUNITY STUDIES

Study	Pb A ($\mu g/m^3$)	Pb S ($\mu g/g$)	Pb HD ($\mu g/g$)	Pb B ($\mu g/dl$)	Estimated Bk ($\mu g/dl$)	Predicted Pb B ($\mu g/dl$)
Idaho	1.0	500	3000	26.7	8.1	26.4
	2.0	1000	3000	28.1		28.9
Los Angeles						
Lancaster	0.64	66	(66)	9.6	4.8	9.7
Los Angeles	6.3	1000	(1000)	14.6		16.4
Toronto						
Control	0.82	99	713	17	6.9	17.2
Smelter	3.01	1715	1550	27		25.1
El Paso						
1977	1.3	427	1479	20.1	6.6	20.3
1972	4.4	1791	22191	31.2		29.4

The regression equation $Pb\ B = Bk\ Pb\ A^{0.03}\ Pb\ S^{0.10}\ Pb\ HD^{0.07}$ was applied to the means of Pb A, Pb S, Pb HD, and Pb B of four epidemiologic studies of children. Idah data are taken from the predicted equation of Yankel et al.[33] with the substitution of the average household dust as cited by Ashley[37]. Toronto data are from Roberts et al The Pb HD cited for El Paso[36] is actually for exterior dust. Los Angeles data[35] equate soil with house dust. Background Pb B (Bk) was estimated by substituting community data at the lower level of Pb A in the Omaha equation. This Bk was then used to predict Pb B at the higher Pb A. Estimated Bk in Idaho was identical to that in Omaha. The Omaha regression predicts Pb B within 2 ug/dl in all 4 studies. (From Angle, C.R., McIntire, M.S. et al: Environmental Lead and Children: The Omaha Study, Journal of Toxicology and Environmental Health, 5:855, 1979 . Used by permission.)

TABLE 5. - PROJECTED EXPOSURE AND NET ABSORPTION OF LEAD IN URBAN CHILDREN

Route	Concentration	Intake per day	Absorbed		Lead Absorbed µg/day
Air	0.75 µg/m^3	10 m^3	40%		3
Water	10 µg/L	1.4 L	50%		7
Food	0.1 µg/g	1 kg	50%		50
Soil/Dust	500 µg/g	0.2 g	17%		150
				Total	210
Paint	10,000 µg/g	0.2 g	17%		340
				Total	550

LEGENDS

Figure 1: Estimate of world wide production of lead over the past 5000 years as related to major historical events (adapted from Patterson[5]).

Figure 2: Concentration of lead in snow pg/g, in samples of inland ice, Greenland, as cited by Herron et al.[13]

Figure 3: Lead concentration µg/gm in bones from ancient Peru (Ericson et al)[15] and Egypt, Nubia and Denmark (Grandjean et al)[16], Britain in the Roman and Anglo Saxon (Waldron et al)[17] eras, contemporary British children (Berry et al)[18] and U.S. adults in the 1950's (Schroeder and Tipton)[19].

Figure 4: The environmental cycling of lead. Lead from industrial emissions enters the atmosphere primarily as inorganic lead, lead halides, sulfates and oxides. Incomplete combustion of tetraethyl lead results in organic lead concentrations that are 10-15% of total atmospheric lead in urban areas, but these are rapidly degraded to the lead oxides found in urban dust and soil.

Figure 5: Map of Omaha showing the commercial (C), mixed commercial-residential (M), and suburban (S) sampling sites and schools as related to three lead emission sources.[27]

Figure 6: Close up of northcentral Omaha showing the percent of substandard housing according to census tracts and the location of 3 lead emission sources. Air sampling was conducted at 3 sites: M (mixed commercial) at the urban high school; C (commercial) and at the urban grade school just south of the battery plant in the center of the area.[25,26]

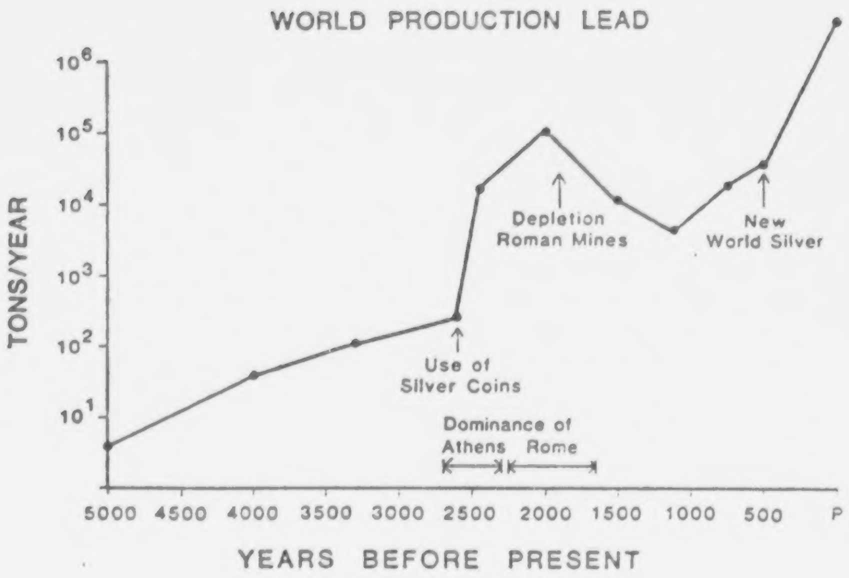
Figure 7: Topographic plot of the blood lead (z axis) of 107 children living in northcentral Omaha with their individual residence plotted on the x and y axes. The thickest black lines define streets carrying more than 3500 vehicles per lane per day. There is a sharp peak of blood leads of children living near the central battery plant and a ridge of increased values paralleling the major north-south street. There is little correlation of blood leads with substandard housing.[25,26]

Figure 8: Blood lead (Pb B, arithmetic means) of 1232 samples from 831 Omaha children, 1-18 years old, 1971-1977.[27]

Figure 9: Blood lead in µg/dl as related to activity of pyrimidine 5'-nucleotidase (P5N) in units defined as the hydrolysis of 1 n mol CMP/min/g hemoglobin, in 15 children.[52,53]

Figure 10: Red cell content of cytidine phosphate (CTP) as related to blood lead. The mean $\pm$ SD of CTP in 13 children with blood lead < 30 µg/dl was 0.22 $\pm$ 0.47 n mol/10^{10} red blood cells. Red blood cells from 11 children with blood lead 30-70 µg/dl had a significantly ($P < 005$) increased content of CTP, with a mean $\pm$ SD of 8.31 $\pm$ 6.21 n mol.[53]

Figure 11: Data from 3 children 2-3 years old treated for an elevated blood lead with EDTA 75 mg/kg/d IM x 4 d. The prompt decrease in blood lead (PbB) was associated with restoration of red cell pyrimidine - 5'-nucleotidase (P5N) to normal. There was no significant change in the red cell content of cytidine triphosphate (CTP) or zinc protoporphyrins (ZPP).[54]



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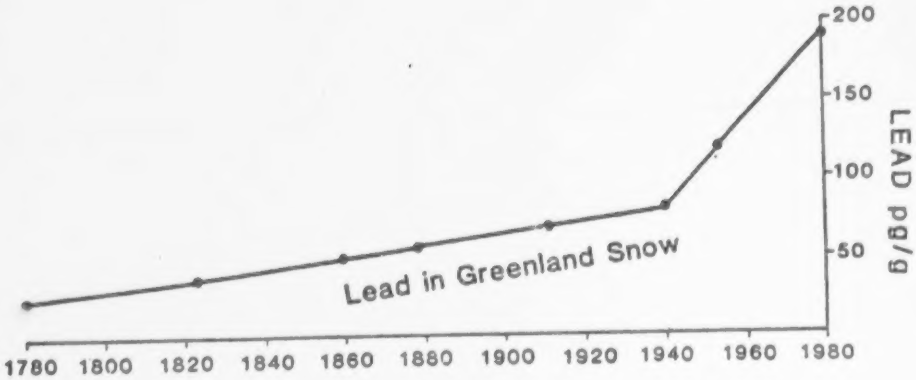

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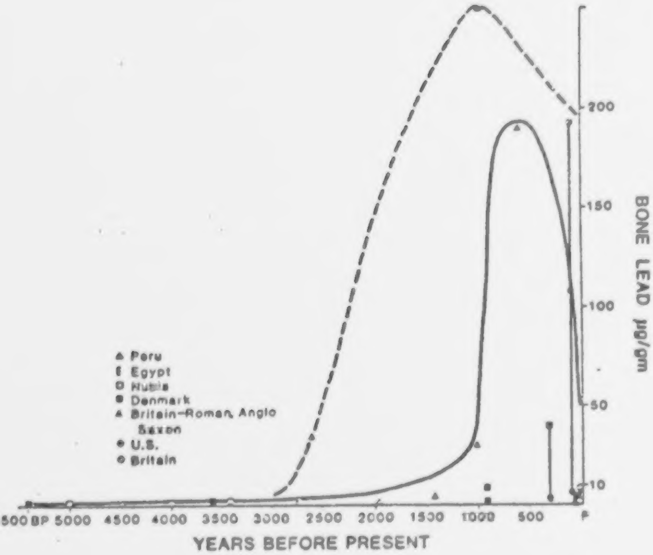

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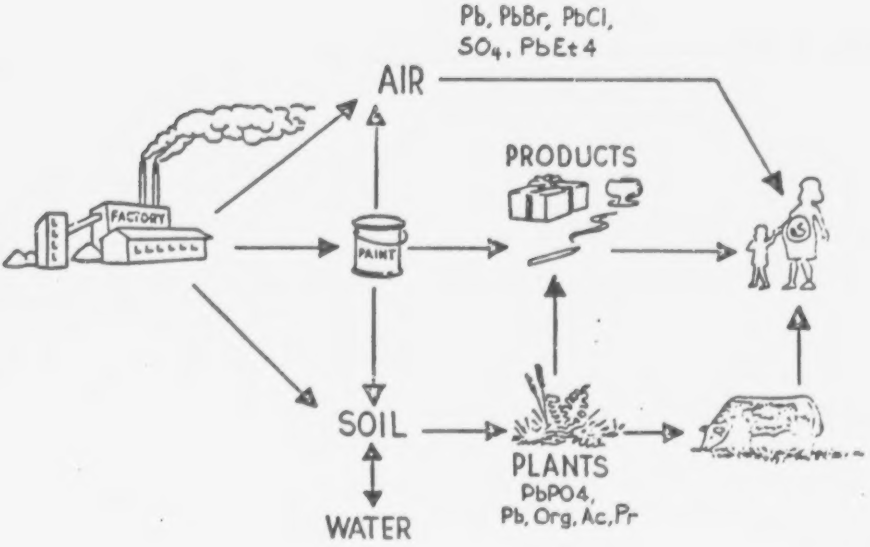
Pb, PbBr, PbCl,
SO4, PbEt4
AIR
PRODUCTS
FACTORY
PAINT
SOIL
PLANTS
PbPO4,
Pb, Org, Ac, Pr
WATER

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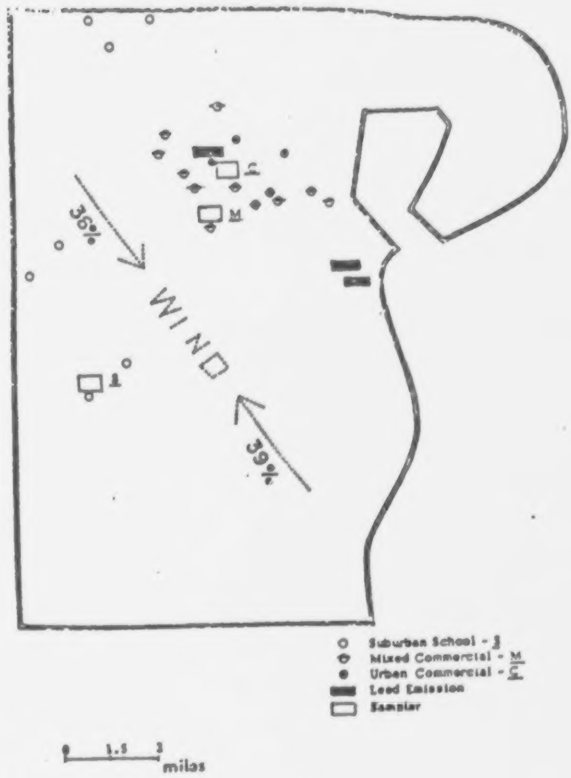
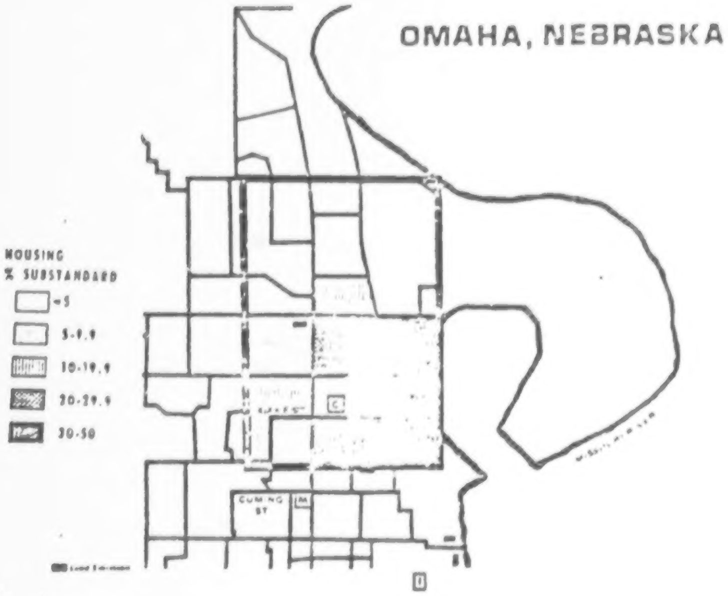

36%
WIND
39%
C
M
S
Suburban School - S
Mixed Commercial - M
Urban Commercial - C
Lead Emission
Sampler
0 1.5 3 miles

Figure 5: Map of Omaha showing the commercial (C), mixed commercial-residential (M), and suburban (S) sampling sites and schools as related to three lead emission sources.[27]


OMAHA, NEBRASKA
HOUSING
% SUBSTANDARD
<5
5-9.9
10-19.9
20-29.9
30-50
C
CUMING ST
M
O

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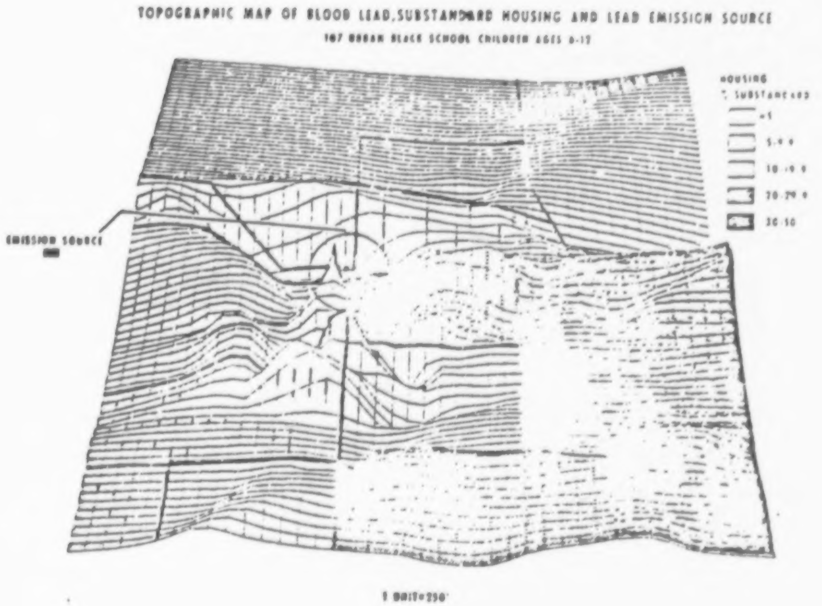

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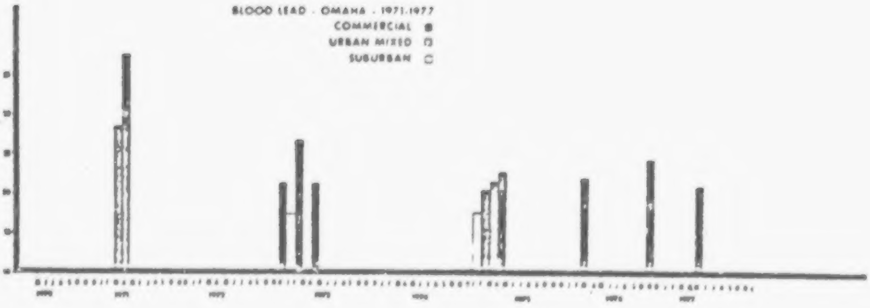

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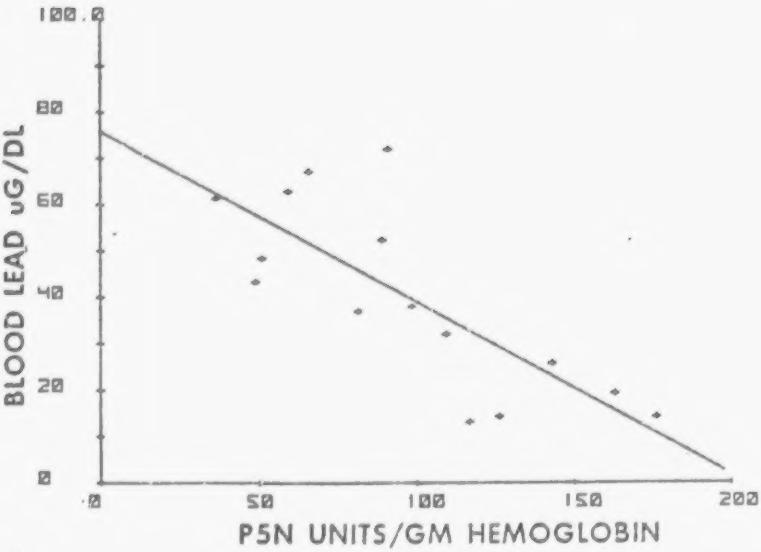

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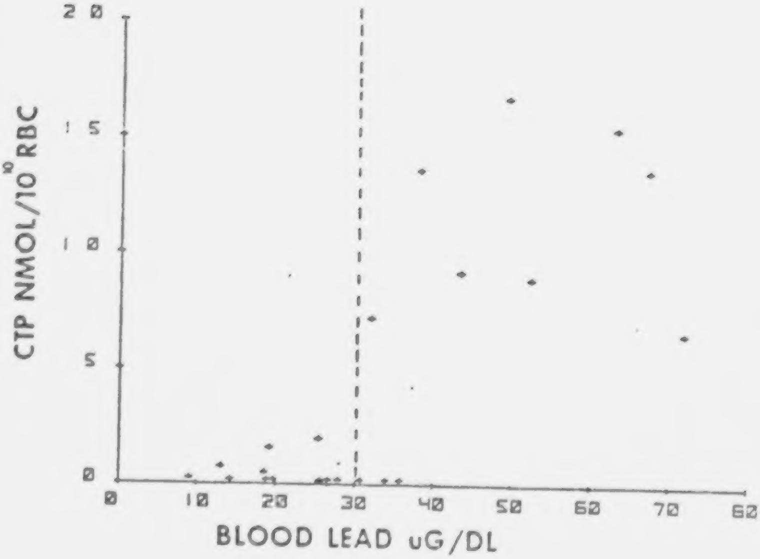

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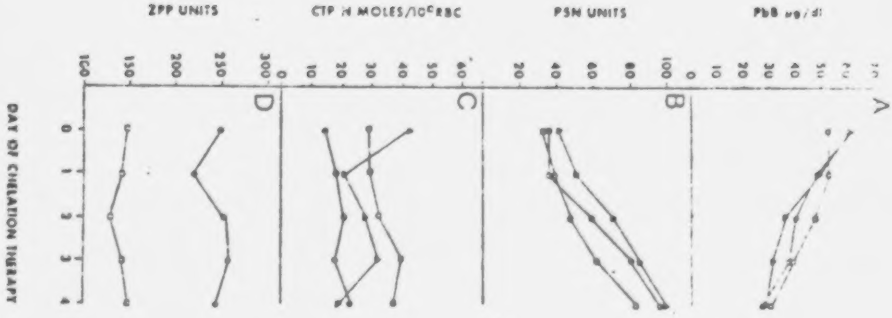

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Exposure to Lead by the Oral and the Pulmonary Routes of Children Living in the Vicinity of a Primary Lead Smelter

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Yearly from 1974 to 1978, a medical survey was carried out among 11-year-old children attending schools situated less than 1 and 2.5 km from a lead smelter. Age-matched control children from a rural and urban area were examined at the same time. The blood lead levels (PbB) of the children living in the smelter area (mainly those attending schools located less than 1 km from the smelter) were higher than those of rural and urban children. The mean PbB levels were usually lower in girls than in boys, especially in the smelter area. Despite a slightly decreasing trend in the annual mean airborne lead concentration at less than 1 km (mean PbA: from 3.8 $\mu g/m^3$ in 1974 to 2.3 $\mu g/m^3$ in 1978) the PbB levels there did not improve, whereas 2.5 km from the plant a significant tendency to normalization of PbB became apparent. Therefore, in the third survey, the medical examination was combined with an environmental study which demonstrated that lead in school-playground dust and in air strongly correlated. Lead on the children's hands (PbH) was also significantly related to lead in air or lead in dust. Less than 1 km from the factory boys and girls had on the average 436 and 244 μg Pb/hand, respectively, vs 17.0 and 11.4 μg Pb/hand for rural boys and girls, respectively. Partial correlations between PbB, PbA, and PbH indicated that in the smelter area the quantitative contribution of PbA to the children's PbB is negligible compared to that of PbH. Thus, the control of airborne lead around the lead smelter is not sufficient to prevent excessive exposure of children to environmental lead. In view of the importance of lead transfer from dust and dirt via hands to the gastrointestinal tract remedial actions should be directed simultaneously against the atmospheric emission of lead by the smelter and against the lead particulates deposited on soil, dust, and dirt.

INTRODUCTION

In 1973, several cases of cattle death around a lead smelter in Belgium drew the attention to environmental pollution by heavy metals. The first environmental surveys within 1 km from the smelter demonstrated levels of lead in air, in soil, and in grass which were at least 10 to 100 times the background values currently found in nonpolluted areas of Belgium being for air 0.23 μg Pb/m^3 (Janssens and Dams, 1975), for soil <25 μg Pb/g (surface layer), and for grass <15 μg Pb/g (dry matter) (Cottenie *et al.*, 1975). Since 1974, the inhabitants and the environment around the plant were extensively surveyed to estimate the biological effects of lead, particularly on children, to pin-point the major lead-emitting sources within the plant and to evaluate the effects of various preventive measures.

Our first medical survey in 1974 (Roels *et al.*, 1976) revealed markedly

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increased—compared to rural children—lead absorption among 11-year-old children attending schools situated less than 1 and 2.5 km from the lead smelter. The undue lead absorption was accompanied by early biochemical indications of disturbed heme biosynthesis. Eighteen months after major improvements had been introduced in the factory to reduce its lead emission, a second medical survey (Roels *et al.*, 1978) was carried out in the same schools (lead smelter and rural areas) on other groups of children which were comparable with those of the first survey. The continuous air monitoring revealed for the smelter area a decrease in airborne lead, but high lead levels in school-playground dust were found at less than 1 km (3000 to 5000 μg Pb/g) and at 2.5 km (about 400 μg Pb/g) compared to the rural school (about 150 μg Pb/g). By comparison with the results of the first survey, a significant tendency to normalization of PbB was found at 2.5 km, but at less than 1 km the biological parameters (particularly PbB, ALAD, FEP) did not improve.

Two hypotheses have been put forward to explain the lack of significant improvements in the biological parameters of the children living less than 1 km from the lead smelter (Roels *et al.*, 1978; Bruaux *et al.*, 1978). Ingestion of lead-containing dust and dirt (probably related to hand contamination) from their surroundings may represent, in addition to air, a supplementary source of increased lead accumulation in these children. The alternative explanation is that a continuous resuspension of dust and dirt particles from the highly contaminated soil around the lead smelter may maintain a very high airborne lead concentration at the breathing height of children. This second hypothesis, however, can be ruled out since a subsequent comparative analysis of air lead levels at 1.6 and 4 m above ground level, performed from June 1977 till September 1978 less than 1 km from the plant, demonstrated that the monthly mean air lead levels at 1.6 m were only 10 to 15% higher than those measured at 4 m as usually.

We have therefore attempted to estimate the relative importance of air and dust on the overall exposure to lead of the children nearest to the smelter. Hence, after the second survey, three more medical surveys (1976, 1977, 1978) were carried out among 11-year-old children living in the smelter area as well as in a rural and an urban area. Between each survey airborne lead (PbA) was continuously monitored and, moreover, during the third survey, hand contamination by lead (PbH) was evaluated in order to study the relationships between PbB, PbA, and PbH.

SUBJECTS AND METHODS

Study populations and their environment. Yearly from 1974 to 1978 a biological monitoring survey was carried out among children living in the vicinity of a large lead smelter (annual lead production about 100,000 metric tons). The first (1974) and the fifth (1978) study were performed at the end of spring (May–June), while the second (1975), the third (1976), and the fourth (1977) were conducted in autumn (mainly October). Each time, a control group of age-matched children from a rural area was examined, except during the fourth survey. The third and the fifth survey also included a group of urban children comparable in age to the lead smelter and rural groups. The overall age for the different groups ranged from 9 to 14 years (mean age 11 to 12 years) and the length of residence at the current dwelling place varied from 0.5 to 14 years (mean 7–10 years). The boys and the girls of the study populations from the lead smelter, the urban (Brussels), and the rural (Herent near Leuven) areas were invariably recruited in the same five schools: one in the urban and rural area and three in the smelter area where two of them (school 1 and 2) are located less than 1 km northeast and the other one 2.5 km east of the plant. A description of the industrialized area and some data reflecting the degree of the environmental pollution (air, soil, grass) around the lead smelter have been published previously (Roels *et al.*, 1976, 1978). Although the lead concentration of suspended particles in the air has been decreasing since 1977, recent studies (Vanderborght and Adams, 1978; IHE, 1979) confirmed that the environmental pollution by lead (and even other heavy metals) remains important, particularly within a distance of 1 km from the high stack: indeed, top-soil (0–10 cm) contained about 2000 to 6000 μg Pb/g, grass about 200 to 800 μg Pb/g dry matter, and in the dwelling areas of the children attending schools 1 and 2, the lead content of the atmospheric dust fallout averaged around 16.4 to 22.0 mg $\mathrm{Pb/m^2/day}$ at 500 m from the high stack, 5.8–7.2 mg $\mathrm{Pb/m^2/day}$ at 700 m, decreasing to about 2 mg $\mathrm{Pb/m^2/day}$ at 1000 m and fluctuating around 0.5–1 mg $\mathrm{Pb/m^2/day}$ at 1.5 km and above. The size distribution of the lead-containing airborne particles in the smelter area demonstrated that particles with a diameter of about 2 μm constituted the most important fraction, while a secondary maximum was found for particles with a diameter between 4 and 8 μm that progressively decreased when the distance to the plant increased from 0.7 to 2.4 km (Degussem *et al.*, 1978). In total, 661 children (328 boys, 333 girls) were examined of whom 214 children (98 boys, 116 girls) attended schools 1 and 2 less than 1 km from the lead smelter, 169 children (115 boys, 54 girls) attended school 3 at 2.5 km from the plant, 55 children (35 boys, 20 girls) lived in the urban and 223 children (80 boys, 143 girls) in the rural area. All the children were in good health and no striking socioeconomic difference was found between the rural, urban, and lead-smelter groups.

Air sampling. Since September 21, 1973, the airborne lead levels 0.7 km (site I) northeast of the 152-m stack have been nearly continuously monitored (IHE, 1977, 1979). The sampler, which is installed on the roof of school 1 at 4 m above the playground, represents the dwelling area of the children attending schools 1 and 2. A second air-sampling station operates 1.2 km (site II) southeast of the high stack and is close to the principal dwelling area of the children attending school 3. By June 21, 1977, a new air sampler 2.5 km east of the high stack was installed 1.6 m above the playground of school 3. However, since the airborne levels at this sampling point were on the average nearly identical with those measured at site II, the air monitoring at 2.5 km was stopped by March 1978. Therefore, the airborne lead concentrations measured at site II were considered also representative for the children attending school 3.

Since June 1977, the air sampling sites I and II were integrated in a network of 13 air-sampling stations operating at different locations within 3.3 km from the high stack. The suspended particles in the air of the smelter zone are collected on Sartorius cellulose nitrate membrane filters (0.45-μm pores) by means of vacuum pumps. The sampling head with the membrane filter is mounted upside down so that the air is aspirated in an upward vertical direction and the filter protected from direct impact of rainfall and atmospheric fallout of heavy particles. About 15

m^3 air is aspirated per day (24-hr samples) through the membrane filter and at 0 hr GMT the vacuum pump is automatically connected to a new filter (IHE, 1977). The air sampling in the school of the urban area (10-hr samples) and the analysis of the filters with the collected particles were performed as previously described for the rural area (Roels *et al.*, 1978). Both methods gave similar results as checked by a comparability test in the urban area.

Dust and dirt. Samples of dust and dirt collected on the children's school playgrounds were analyzed for lead as described previously (Roels *et al.*, 1978). The samples were collected at the time the hand contamination by heavy metals was determined in the course of the third survey.

Contamination of hands. In order to estimate the contamination of the children's hands by trace metals (lead, cadmium, manganese, arsenic), 500 ml of HNO_3 0.1 N was poured slowly over the palm of one hand (dominant hand) while the fingers are slightly spread. The diluted nitric acid was collected in a metal-free polyethylene bottle (500 ml) by means of a metal-free polyethylene funnel. After each "hand-rinsing," the funnel was rinsed with 500 ml HNO_3 0.1 N. Bottle and funnel blanks were taken in each school before and after the "hand-rinsing" operations which took place in the afternoon (between 2 and 4 PM) on sunny days. The children had not been told before that their hand contamination would be evaluated.

The heavy metal concentrations in the diluted nitric acid "hand-rinsings" were measured by electrothermal atomic absorption spectrophotometry using Perkin-Elmer instruments, Model 305, 360, or 420, equipped with a deuterium background corrector, a HGA-74 or HGA-76B graphite furnace (Perkin-Elmer), and an automatic sampling device Perkin-Elmer Model AS-1 for the injection of 20-μl aliquots. Appropriate dilutions and standard curves in HNO_3 0.1 N (except for arsenic, see: Buchet *et al.*, 1980) were run and the amounts of the metals are expressed in micrograms per hand.

Blood analysis. A sample of blood was taken from each child by venipuncture using standardized disposable syringes and hypodermic needles free from heavy metal contamination. Aliquots of 5 ml blood were immediately transferred into two heparinized metal-free polyethylene or polystyrene tubes for blood lead analysis and for routine hematological measurements including hematocrit, hemoglobin, and blood cell counts with a Hemalog 8 (Technicon). The remainder of the blood sample was kept in a glass tube to separate serum for determination of its iron level (except first survey). The blood samples were kept at 4°C and all the analyses were performed within 24 hr. From the examination of the hematological data, it could be concluded that none of the children suffered from iron deficiency anemia.

Throughout the five surveys electrothermal atomic absorption spectrophotometric methods were used for blood lead analysis. The blood lead of the children was determined over the whole study period independently by the laboratory of the Ministry of Public Health and our laboratory: the average blood lead levels reported by these laboratories for each population group were very close and usually differed less than 10%. Moreover, they participated regularly in quality control programs organized on national or European scale in order to ascertain their proficiency in measuring blood lead.

Statistical analysis. The statistical difference between subgroups was ascertained by one-way variance analysis. Pearson's and Spearman's correlation coefficients were also calculated.

RESULTS

Correlation between Air Lead and Blood Lead: An Indirect Relationship

An overview of the mean airborne and blood lead levels corresponding to the several children population groups examined during the five surveys is presented in Table 1. The reported airborne lead levels are the averages of the monthly mean values observed during the 9 months preceding the first survey and during the time interval between the following surveys. Hence, the PbA values given in Table 1 are somewhat different from the calendar year mean values: this applies particularly to the sampling station located less than 1 km from the lead smelter where mean PbA amounted to 3.79 μg Pb/m^3 in 1974, 3.32 μg Pb/m^3 in 1975, 3.59 μg Pb/m^3 in 1976, 2.84 μg Pb/m^3 in 1977, and 2.27 μg Pb/m^3 in 1978. Since 1977 the airborne lead concentrations in the dwelling areas of the children attending the school at 2.5 km are comparable with those recorded in the urban school. Most likely due to less motor vehicle traffic in the vicinity of the rural school the airborne lead level there is lower than in the urban school.

Table 1 indicates that the mean PbB level is usually lower in girls than in boys, particularly in the smelter area (the highest individual PbB value recorded was 49 μg/100 ml). Less than 1 km from the lead smelter no improvement in the mean

TABLE 1

MEAN AIRBORNE AND BLOOD LEAD LEVELS RECORDED DURING FIVE DISTINCT SURVEYS (1974 TO 1978) FOR STUDY POPULATIONS OF 11-YEAR OLD CHILDREN LIVING LESS THAN 1 km OR 2.5 km FROM A LEAD SMELTER, OR LIVING IN A RURAL OR URBAN AREA

Study populations		Pb−Air (μg/m^3)	Blood lead concentration (μg/100 ml)					
			Total population		Boys		Girls	
			n	Mean ± SD	*n*	Mean ± SD	*n*	Mean ± SD
1 Survey (1974)	<1 km	4.06	37	30.1 ± 5.7	14	31.0 ± 5.5	23	29.6 ± 5.9
	2.5 km	1.00	—	—	14	21.1 ± 3.4	—	—
	Rural	0.29	92	9.4 ± 2.1	28	9.7 ± 1.6	64	9.3 ± 2.2
2 Survey (1975)	<1 km	2.94	40	26.4 ± 7.3	19	27.4 ± 6.5	21	25.4 ± 8.1
	2.5 km	0.74	29	13.6 ± 3.3	17	14.8 ± 3.6	12	11.9 ± 1.9
	Rural	0.31	45	9.1 ± 3.1	14	8.2 ± 2.1	31	9.5 ± 3.4
3 Survey (1976)	<1 km	3.67	38	24.6 ± 8.7	18	28.7 ± 8.0	20	20.8 ± 7.6
	2.5 km	0.80	40	13.3 ± 4.4	24	15.6 ± 2.9	16	9.8 ± 3.8
	Urban	0.45	26	10.4 ± 2.0	17	10.6 ± 2.0	9	9.9 ± 2.0
	Rural	0.30	44	9.0 ± 2.0	21	9.2 ± 2.3	23	8.7 ± 1.7
4 Survey (1977)	<1 km	3.42	56	28.9 ± 6.5	27	31.7 ± 9.5	29	26.4 ± 8.7
	2.5 km	0.49	50	14.8 ± 4.7	34	15.7 ± 4.8	16	13.0 ± 4.3
5 Survey (1978)	<1 km	2.68	43	27.8 ± 9.3	20	29.3 ± 9.8	23	26.5 ± 8.9
	2.5 km	0.54	36	16.0 ± 3.8	26	16.6 ± 3.5	10	14.3 ± 4.2
	Urban	0.56	29	12.7 ± 3.1	18	13.4 ± 2.3	11	11.5 ± 4.0
	Rural	0.37	42	10.7 ± 2.8	17	11.9 ± 3.0	25	10.0 ± 2.4

blood lead concentration was observed throughout the 5-year observation period. On the other hand, the mean PbB levels of the children attending the school 2.5 km from the lead smelter never exceeded 20 μg/100 ml since 1975, although the PbB values were still higher than those measured in the urban and rural schools, particularly for the boys. The two surveys among urban children demonstrated for boys as well as for girls slightly higher PbB values than for the corresponding rural groups. The apparent relationships between the mean blood lead levels (total population, boys, girls) and lead in air are presented in Fig. 1. Similar correlations were obtained whether parametric (Pearson) or nonparametric (Spearman) statistics were used. These results tend to indicate that when Pb–air increases by 1 μg/m^3 PbB would increase by approximately 5 μg/100 ml. This figure, however, largely exceeds the fractional contribution of Pb–air to Pb–blood which is estimated in the range 1 to 2 μg Pb/100 g blood per 1 μg Pb/m^3 on the basis of experimental and epidemiological results principally obtained on adults (Mahaffey, 1977). According to Chamberlain *et al.* (1978), there is conflicting evidence as to whether the effect of Pb–air on Pb–blood is greater for children than for adults. Analysis of literature data by these authors points to little difference, although epidemiological studies (Johnson *et al.*, 1975; Tsuchiya *et al.* 1977) suggest a more important effect in children, possibly connected with indirect uptake of lead from other sources correlated with Pb–air. A similar indirect mechanism (probably hygienic) may explain differences observed in blood lead levels between boys and girls (Table 1). Indeed, since boys and girls of 11–12 years old have almost similar respiratory ventilation (Geigy Scientific Tables, 1973) another cause associated with Pb–air must be invoked to explain the greater apparent fractional contribution of Pb–air to Pb–blood in boys than in girls (Fig. 1).

Interrelationships between Pb–Air, Pb–Dust, and Pb–Hand

A simple extrapolation of the relationship found between air lead and blood lead in urban and suburban situations may not be valid in heavily contaminated areas

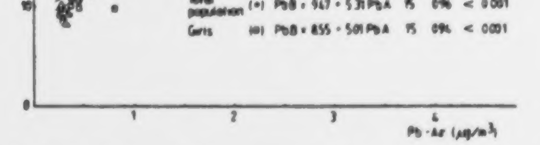

FIG. 1. Apparent relationship between the mean concentrations of lead in blood and lead in air as observed for the distinct groups of 11-year-old children examined from 1974 to 1978.

near lead smelters, where fallout of lead and high lead levels in dust may contribute significantly to the overall exposure. Therefore, in order to pin-point the most important factor contributing to the blood lead of children in the smelter area, an environmental survey (air, dust, lead on hand) coupled with blood lead analysis was carried out in 1976 (Table 1, Fig. 2). The rural air lead level of 0.30 µg/m³ was the lowest of the four geographic areas. The urban air lead concentration amounted to a somewhat higher value of 0.45 µg/m³, whereas 0.80 and 3.67 µg/m³ were measured, respectively, 2.5 km and less than 1 km from the lead smelter. Lead concentration of school-playground dust was similar in the urban and rural area, namely 112 and 114 µg/g, respectively, but was about four times higher in the school at 2.5 km (466 µg/g) and even more than 20 times in the schools less than 1 km (2560 µg/g) from the lead smelter. The average amounts of lead found on the hands of the children from the distinct areas followed the air and dust lead levels characteristic for the children's environment. Boys were found to have invariably higher amounts of lead (µg/hand) on their hands than girls: the rural school, boys 17.0 µg vs 11.4 µg for girls; the urban school, boys 20.4 µg vs 12.7 µg for girls; the school 2.5 km from the lead smelter,

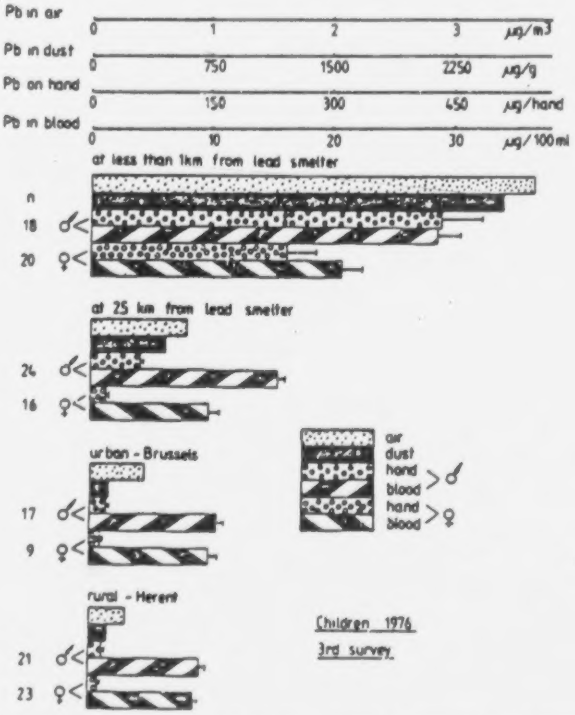

FIG. 2. Lead in air, in dust, in blood, and on hand found during the third survey (1976) among 11-year-old children living less than 1 or 2.5 km from the lead smelter, or living in an urban or rural area.

boys 62.2 μg vs 20.0 μg for girls; and schools less than 1 km from the lead smelter, boys 436 μg vs 244 μg for girls.

These environmental parameters are interrelated. Lead in dust (PbD) is highly correlated with lead in air ($r = 0.999$, $P < 0.001$; $PbD = -151.4 + 739.0\ PbA$). Lead on hand is also significantly related to lead in air or lead in dust showing moreover significantly higher ($P < 0.001$) slopes for boys than for girls (Table 2). The multiple regression coefficients of PbH · PbA · PbD are $R = 0.999$ and $R = 0.995$ for boys and girls, respectively. The higher degree of hand-cleanliness in girls than in boys probably results from differences in their everyday activities.

Interrelationships between Pb–Air, Pb–Hand, and Pb–Blood

Yankel *et al.* (1977) deduced from a multiple regression analysis that five factors significantly influenced the probability of a child experiencing an excess in blood lead level: (1) ambient air lead concentration, (2) lead concentration of the soil, (3) age of the child, (4) cleanliness of the home, (5) occupation of the parents. In our study, the third factor cannot be considered a variable influencing PbB since children within a definite age range were chosen. The fifth factor is of little importance to our study, since there were no major socioeconomic differences between the various study populations. Only 5 children out of 78 lead-smelter children examined during the third survey had their father employed at the smelter. Their PbB values (11.7, 14.2, 18.0, 18.7, and 30 μg/100 ml, respectively) were in the same range as those of the other children living in the same area. This may be due to the fact that in Belgium, workers are not allowed to return home with their work clothes.

The population of the smelter area was also aware of the environmental lead pollution and was advised since 1973 to avoid consumption of home-grown vegetables and fruit. The drinking water in the four geographical areas did not contain more than 0.05 mg Pb/liter. Taking into account these conditions, the assumption can be made that the PbB level of the children primarily reflects uptake by inhalation of airborne lead and by ingestion of lead from hands contaminated through lead fallout and dust (in our age groups pica did not occur, however, nail-biting and finger-sucking was still frequently observed).

As for lead on hands, the average blood lead levels of the boys in the four distinct geographical areas were also consistently higher than those of the corresponding groups of girls as shown in Table 1 (see third Survey, 1976) and Fig. 2.

TABLE 2

INTERRELATIONSHIPS BETWEEN LEAD IN DUST (PbD) OR LEAD IN AIR (PbA) AND LEAD ON HAND (PbH) AS FOUND IN THE GROUPS OF BOYS AND GIRLS EXAMINED DURING THE THIRD SURVEY (1976)

Population groups	Number	Correlated parameters[a]	r	P	Regression equation
Boys	4	PbH · PbA	0.999	< 0.001	$PbH = -32.1 + 127.2\ PbA$
		PbH · PbD	0.999	< 0.001	$PbH = -5.9 + 0.172\ PbD$
Girls	4	PbH · PbA	0.995	< 0.005	$PbH = -21.8 + 71.8\ PbA$
		PbH · PbD	0.994	< 0.010	$PbH = -6.8 + 0.097\ PbD$

[a] The parameters are expressed in μg Pb/g for PbD, in μg Pb/m^3 for PbA, and in μg Pb/hand for PbH.

For the lead-smelter groups the differences in blood lead between boys and girls were even highly significant ($P < 0.001$ at 2.5 km and $P < 0.005$ at less than 1 km) and paralleled the differences in lead on hand found between boys and girls in each of these two groups. It is interesting to notice that, during the third survey, only the girls at less than 1 km from the plant demonstrated an elevated mean PbB value, whereas the other three groups of girls (at 2.5 km, urban, rural), in spite of a different air lead, exhibited low and similar PbB values. This could be related to their low hand contamination showing relatively small difference among these groups. However, the mean PbB values in the rural and urban boys were clearly different from the mean PbB value found in the boys attending the school 2.5 km from the lead smelter. The quantity of lead on the hands of these boys amounted to about 60 μg/hand and is three times the quantity found for rural or urban boys and for girls attending the same school 2.5 km from the factory. This might suggest that lead ingestion from hand contamination significantly influences blood lead concentration when lead on hand becomes higher than 20 μg/hand. In order to examine the interrelationship between lead in blood, lead in air, and lead on hand we assumed as many other authors a linear relation between PbB and PbA, but a curvilinear relation between PbB and PbH since recent studies on the relation between water lead and blood lead (Lauwerys *et al.*, 1977; Moore *et al.*, 1977) suggest a nonlinear relationship between oral lead intake and blood lead. The multiple regression coefficient of PbB · PbA · log PbH, calculated for the four geographic groups of boys and girls combined ($n = 8$), is highly significant ($R = 0.976$; $P < 0.001$). Standardization for PbA resulted in a still significant partial correlation between PbB and log PbH, whereas the partial correlation coefficient between PbB and PbA becomes insignificant when standardized for log PbH (Table 3). This means that ingestion of lead from lead-contaminated hands is a much more important factor contributing to PbB than does PbA. That the influence of PbA on the relationship PbB · log PbH appears to be very weak in our study populations is emphasized by the fact that the regression equation of the simple correlation between PbB and log PbH (Table 3) has practically a similar slope and intercept as the partial regression equation neglecting the partial contribution of PbA to PbB. According to this finding, it is possible to draw a simple correlation regression line for the relationship PbB · log PbH as calculated for the combined groups ($n = 8$) of boys and girls (Fig. 3).

Of course, due to the fact that PbA strongly correlates with PbH, very significant indirect correlations between PbB and PbA are to be expected for boys ($r = 0.983$, $P < 0.025$) and for girls ($r = 0.995$, $P < 0.005$). However, from the results discussed above, we know that these correlations are primarily due to the influence of PbH on PbB. This leads to an erroneous estimate of the fractional contribution of PbA to PbB as suggested by Fig. 1. Thus, it may not be concluded that in this study PbB is directly related to PbA in terms of a cause and effect relationship.

DISCUSSION

Increased lead concentrations in the ambient air of large cities mostly due to vehicle emissions—and near lead smelters have been the subject of concern because of the possible health effects particularly in children (NAS, 1972; Lin-Fu,

TABLE 3

RELATIONSHIP BETWEEN LEAD IN BLOOD (PbB), LEAD IN AIR (PbA), AND LEAD ON HAND (PbH) AS FOUND IN THE COMBINED ($n = 8$) GROUPS OF BOYS AND GIRLS EXAMINED DURING THE THIRD SURVEY (1976)

Simple and partial correlation				
Correlated parameters[a]	Constant parameter	r	P	Regression equation
PbB–PbA	log PbH	0.002	>0.05	PbB = −4.27 + 11.50 log PbH +0.007 PbA[b]
PbB–log PbH	PbA	0.810	<0.025	
PbB–log PbH	—	0.976	≤ 0.001	PbB = −4.33 + 11.54 log PbH

[a] The parameters are expressed in μg Pb/100 ml for PbB, in μg Pb/m³ for PbA, and in μg Pb/hand for PbH.

[b] The partial contribution of PbA to PbB has to be neglected ($r = 0.002$, not significant).

1973, 1975; Roberts *et al.*, 1974a; Landrigan *et al.*, 1975a, 1976; Roels *et al.*, 1976, 1978; Tsuchiya *et al.*, 1977). However, lead in ambient air which increases lead concentration in dust due to fallout has been suspected to cause a considerable enhancement in the daily oral lead intake of children who developed increased blood lead in those areas (NAS, 1972; Sayre *et al.*, 1974; Lepow *et al.*, 1975; Day *et al.*, 1975; Yankel *et al.*, 1977; Roels *et al.*, 1978).

Angle *et al.* (1974) concluded from an environmental study in Omaha, that blood lead of urban school children, all past the age of pica, showed significant correlations with exterior "fallout" lead, but little or no correlation with air lead. Roberts *et al.* (1974b) stated that the major route of lead absorption by children living in different areas of Toronto contaminated by secondary lead smelters appeared to be ingestion of dirt and dust rather than inhalation of suspended particles. Around the El Paso ore-smelter it appeared that the more mobile fractions (air, dust) of environmental particulate lead, rather than soil lead, were those most closely associated with human uptake (Landrigan *et al.*, 1975b).

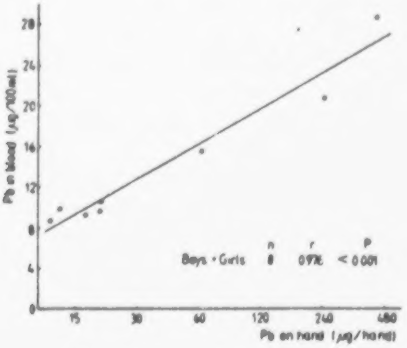

FIG. 3. Relationship between the mean concentration of lead in blood and on hand as observed for the combined ($n = 8$) groups of boys and girls examined during the third survey (1976).

Unlike many North American cities where ingestion of lead-based peeling paint is considered an important cause of excessive lead absorption (Lin-Fu, 1972), in Belgium this kind of lead problem does not exist. Therefore, the present paper deals with environmental lead pollution (air, dust) which in the smelter area is mainly due to airborne lead-containing effluents from the plant (fumes from stack and smelting-ovens, ore dust) and in the other areas to combustion of leaded gasoline.

The study of Yankel *et al.* (1977) among children near a lead smelter in Kellogg, Idaho, is actually one of the most extensive environmental analysis providing a relationship—based on epidemiological data—to predict blood lead from given air lead concentrations in an environment with excessive lead contamination. PbA (ranging up to 17 μg/m^3) and lead in surface soil (up to 7500 ppm) were found to be the most significant factors. Yankel *et al.* (1977) calculated that the blood lead contribution from a 1 μg Pb/m^3 increment of air lead exposure would amount for children to about 1.1 – 1.4 μg Pb/100 ml blood over a range of ambient air lead exposure of 1 to 5 μg/m^3, while lead in soil up to 5000 μg/g would rather weakly influence PbB (cf. also Landrigan *et al.*, 1975b; Barltrop, 1975).

The environmental lead contamination in the surroundings of the Kellogg and our lead smelter seemed to be comparable. Although the lead concentration of air and surface soil around the Kellogg smelter appears to be somewhat higher, particularly for air, we assumed that Yankel's relationship between childhood PbB levels and environmental lead exposure could be applied to our school children. Accordingly, the difference in PbA levels (first survey) between the rural and lead smelter (less than 1 km) areas would predict for the lead smelter children a mean PbB level at the most 5.3 μg/100 ml higher than that of the rural children; for the fifth survey, the PbB enhancement would be no more than 3.3 μg/100 ml. The discrepancy between the predicted PbB value—which would never exceed 15 μg/100 ml—and the measured mean PbB levels of 25 to 30 μg/100 ml strongly suggests an additional pathway of lead intake near the lead smelter. As mentioned above, lead of industrial origin transported home from the workplace is not likely to play an important role. Since pica did not occur in our study populations, it must be inferred that lead particulates deposited by the smelter in the environment are ingested mainly via excessively lead-contaminated hands. This lead exposure mechanism is indirectly supported by the finding that the PbB levels in nine teachers of the schools less than 1 km from the lead smelter were in the normal range (mean ± SD, 18.0 ± 1.3 μg/100 ml; range, 15.1 – 19.5 μg/100 ml), although significantly higher than those observed in nine teachers of the rural and urban schools (mean ± SD, 12.8 ± 3.3 μg/100 ml, range 8.6 – 18.4 μg/100 ml). The difference in blood lead between these two groups of adults more or less reflects the difference in air lead as calculated according to Yankel *et al.* (1977). It seems thus that the principal route of lead absorption differs greatly between children and adults. Our results suggest that inhalation of airborne lead constitutes by far the most important source of a slightly increased PbB in adults from the smelter area. In children of this area, however, a considerably enhanced oral daily intake of lead via soiled hands seems to be the main factor explaining their increased PbB. Indeed, the dose-effect relationship between log PbH and PbB (Table 3) would—irrespective of PbA—predict for a PbH = 10 μg/hand (like in the rural area) a PbB of about 7.2 μg/100 ml and for a PbH of 50, 100, 500, and 1000 μg/hand PbB of 15.3, 18.8, 26.8, and 30.3 μg/100 ml, respectively. In the present study, the quantitative contribution of PbA to PbB was found to be rather negligible compared to that of PbH. That lead on hands appears to be a useful environmental indicator of the degree of lead ingestion is also supported by the fact that the higher PbH level for boys than for girls are closely reflected in their respective PbB levels, even in the rural and urban areas.

Yankel *et al.* (1977) considered only in the third place lead containing dust as a possible source of exposure to environmental lead. They observed a fairly weak correlation with PbB ($r = 0.21$), but this is most likely to be attributed to their less quantitative and rather subjective assessment of cleanliness of the homes simply in terms of clean, moderate, or dusty. Furthermore, the lack of a significant correlation between PbB and the lead concentration in house dust samples obtained from vacuum cleaner bags is not surprising, because it is not the concentration but the absolute amount of lead recovered as dust per surface unit which is related to the cleanliness of the houses. In our present study, we attempted to assess objectively the factor cleanliness related as close as possible to ingestion of environmental lead. This may explain the very strong correlation between blood lead and hand cleanliness which provides a quantitative basis for assessing to what extent lead ingestion from contaminated hands may contribute to blood lead levels in children living around a lead smelter.

CONCLUSION

The overall influence of environmental lead contamination (suspended particles, dust fallout, and pollution of surface soil) on children living around a lead smelter should take into account their physiological characteristics and habits. Therefore, the environmental control strategy should be directed to all routes of exposure bringing about significantly enhanced lead absorption. Measures aiming only at the decrease of airborne lead inhalation would be to a large extent insufficient in the smelter area. Since intake of lead from contaminated hands roughly contributes to the children's PbB by at least with a factor of 2 to 4 more than does air lead, it can be stated that the actual airborne lead concentrations in the smelter area does not represent as much an indicator of exposure as does lead contamination of hands. Therefore, in a lead smelter area, the enforcement of a permissible limit for airborne lead alone may not necessarily prevent an excessive exposure of children to environmental lead, since past emission of lead and possibly direct transfer of lead-containing dirt (e.g., through road transport) will maintain a high level of lead in soil-dust and dirt irrespective of the current concentrations of atmospheric lead.

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Investigations into Sources of Lead in the Environment of Urban Children

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The lead content of air, house dust, dirt, dirt on hands, food, and water was investigated in order to ascertain the cause of the chronically elevated blood lead levels in ten 2–6 year old Hartford children. Recent ingestion of leaded paint had been eliminated as a significant factor.

The mean ambient air levels at 2 and 5 ft. from the ground where the children played varied from 1.7–7 $\mu g/m^3$.

On the basis of "mouthing" behavior observed among the ten subject children, the mean lead levels in dirt (1200) $\mu g/g$) and dust (11,000 $\mu g/g$) and the mean lead concentration of lead on hands (2400) $\mu g/g$), it is likely that the "hands in mouth" exposure route is the principal cause of the excessive lead accumulation in these children.

INTRODUCTION

Epidemiologic studies of lead poisoning in children have consistently implicated the abnormal ingestion of lead-based peeling paint as the primary etiologic factor. (Lin-Fu, 1972) Additional evidence, however, indicates that other environmental lead sources, such as the ingestion of lead-contaminated dirt and dust by children with pica (ingestion or "mouthing" of nonfood items), might be involved in the development and maintenance of elevated blood lead levels, espe-

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cially the "borderline elevations" (40–60 $\mu g\%$) found in a considerable percentage of urban children. (Guinee, 1971)

In 1971, 272 preschool urban children and 230 rural children of similar age were screened for abnormal lead exposure(Cohen, 1973). Eighteen of the 19 rural children found to have excessive lead body burdens, defined as blood lead of $\geqslant 40\ \mu g\%$, (Statement of Surgeon General, 1971) resided in homes containing at least one accessible surface with $\geqslant 1\%$ lead paint. On the other hand, 75% of the urban children with similarly elevated lead burdens were found to have a similar paint hazard on accessible indoor and exterior home surfaces. In addition, over 50% of the screened urban children had blood leads above 30 $\mu g\%$ compared with less than 10% of the rural children. Similar findings have been reported by Sayre, 1974 and co-workers in Rochester, New York. It appears that urban youngsters are more excessively exposed to lead than their rural counterparts. It is difficult to implicate a single lead source for this urban group since even the occasional ingestion of small leaded paint chips would be expected to exceed the maximum permissable daily lead intake resulting in elevations of $> 60\ \mu g\%$ blood lead (King, 1971).

Attention has been recently focused on lead in air, introduced primarily from the combustion of leaded gasoline, as a significant source, since inner city children often live and play in close proximity to moving traffic. Darrow (1973) has shown that atmospheric levels of lead may average 8 $\mu g/m^3$ at or below 4 ft. from the ground. It has been estimated by Kehoe, (1961) that 30–50% of lead in inspired air may ultimately be retained by the body.

Fallout of lead from the air is believed to contribute significantly to the high lead content found in urban soil (Hunt, 1971) and house dust (Sayre, 1974). Children with pica or excessive mouthing of nonfoods, who play in such contaminated environments, can potentially ingest significant quantities of lead from these environmental sources. Foods which are grown in soil, as well as meats, fish, and poultry, may have variable lead levels depending upon the natural lead content of the soil as well as fallout of airborne lead (Barltrop, 1973). Likewise, water may naturally have increased lead content or may be contaminated by flow through leaded pipes or through contamination of our waterways so that the acceptable concentration of 50 parts per billion is exceeded.[1]

The present investigation was undertaken during June–August, 1973, to identify the environmental and behavioral factors in ten preschool children with lead levels $\geqslant 40\ \mu g\%$ for 6–25 months. Readily accessible leaded paint had been eliminated as a significant source for at least 3 months for some, to over a year for most.

MATERIALS AND METHODS

Description of the Population

Five children were black and the rest were Puerto Rican. Elevated body lead burdens had been detected as a result of community or hospital screening programs, although only one had significant symptoms or signs of lead intoxication. All had been under medical observation for more than 6 months at the time that the study was initiated. The mean age was 52 months, and the blood lead levels were 40–70 $\mu g\%$. Several of the children had reached peak blood lead levels of 70–120 $\mu g\%$, and half had received at least one course of chelation therapy when blood lead levels had exceeded 60 $\mu g\%$. All but one had a previous history of pica, and current pica was reported for four.

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[1] U. S. Department of Health, Education and Welfare, Public Health Service. Drinking water standards, Public Health Service, Pub. No. 956 (revised), 1962. Washington, D. C., 61 pp.

Housing

Eight of the ten presently lived in homes built prior to 1940, and two had moved within a year to a 10 year old urban public housing project with little traffic. The mean length of time in the present housing was 3 years for the entire group.

All dwellings occupied by the families of these children within the 12 months preceding the present study, as well as those where the children were frequent visitors, were inspected by the Hartford Health Department Housing Code Enforcement Division. Lead in excess of 1% concentration in indoor flaking paint or plaster had been eliminated, or the family was relocated. Exterior leaded surfaces in poor repair and accessible to the children had been identified, repairs recommended, and families educated about keeping children away from dangerous areas. Compliance had been good.

Plan of Study

The environmental samples obtained for analysis for lead concentration included air, soil, house dust, hand soil, food, water, and nail polish.

Air Sampling Procedure

Air was sampled[2] in front of four of the residences presently occupied by families of study children and at a single suburban site. Two-hour samples at 8:00–10:00 AM or 3:00–5:00 PM were obtained at each site on five or six nearly consecutive days when there was no rain. The sampler was placed 10–15 ft from the street where children played, with the filter holders pointed toward the street. The filter holders were set at 2 and 5 ft from the ground, using one filter in each holder for each 2 hour sample. Used filters were stored in a plastic petri dish and delivered to the laboratory within 2–3 days.

Surface Soil, Dust, Hand Dirt Collection

Surface soil and dust samples were obtained from residences presently or recently occupied by the ten study children. At each residence, three to ten samples each of dirt and dust were obtained. Soil samples were taken from the areas where children were known or likely to play, usually more than 10 ft. from the dwelling. Dust samples were collected from a number of indoor sites

[2] The sampling apparatus consisted of an air pump with two vacuum gauges connected to the two gas meters to measure air flow, which were connected via rubber tubing to the two millipore filter holders set on a wooden stand at 2 and 5 ft. Indoor outlets and a portable alternator, operated on lead-free gasoline, served as sources of electricity. Gelman GA-4 membrane filters (pore size 0.8 m, 47 mm in diameter), judged to be about 100% efficient for trapping of all lead particles, were used.

although floors and windowsills were favored. Samples of dirt from hands of study children were also obtained in the same manner during the course of play around the dwellings where they lived. Comparable samples of air dust and dirt were obtained from six residences on the same streets where children of similar ages known to have blood levels of < 40 $\mu g\%$ resided. These six children provided hand samples. These will be designated as control samples.

Method of Collection of Dust, Dirt, and Hand Samples

The technique described by Darrow, (1973) was used to collect dust, dirt and hand samples. Preweighed self-adhesive labels[3] were employed. A label was usually pressed once on a single area, though it was often necessary to press several times on a given area to obtain an adequate sample. Pebbles and paint chips were avoided or removed. A single sample was obtained from each site. The sample was placed in a wax bag, stored away from the sun to prevent dehydration, and delivered to the laboratory within 2–3 days. Information was kept concerning the exact location of the sample. Any sample reported to have paint chips by either the investigator or the laboratory was excluded. None of the hand samples contained paint chips.

Testing of Tap Water in the Dwelling Units of Study Children

In August 1973, a staff member from the Environmental Health Services Division of the Connecticut State Department of Health obtained water samples from faucets in 14 dwelling units, presently or recently occupied by families of study children, which were analyzed for lead.

Food and Nail Polish Samples

With informed parental consent, three to six 1 hour home visits were made by one of the authors (MBG) to make observations of the mouthing behavior of study children. She also noted brands of canned foods and beverages used by the study children. At the end of the study period, a list was compiled of 18 of the most frequently employed foods and beverages and four brands of nail polish. Samples of each of these were purchased from local grocery stores and submitted unopened to the laboratory for analysis of lead content.

Observations of "Mouthing Behavior"

The number of times that children put hands or nonfood objects in their mouths was recorded during the observation period. A score of 1–4 was assigned to describe the behavior patterns observed. A score of 1 meant that no abnormal mouthing was observed, and 2 indicated that occasionally the child was seen with hands or nonfoods in the mouth. A score of 3 represented moderate frequency, and 4 indicated that the child was seen with hands or nonfood items in the mouth 50% or more of the time. Although it would have been desirable, it was not possible to make similar observations of the six control children.

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[3] Avery #5615, 1/16 × 3½ in., 21.5 cm².

Laboratory Analyses

All environmental and food samples were tested for lead at the Connecticut State Health Department, Environmental Chemistry Laboratory. Atomic absorption analysis was employed using a Varian Techtron Atomic Absorption Spectophotometer. Standard solutions of six different concentrations were used to establish a working curve with each set of samples. The minimum sensitivity was 0.15 mg/ml.

The labels were equilibrated in a dessicant cabinet for a minimum of 24 hours before the preuse weight was obtained. Upon receipt of the sample, the label was again equilibrated for 24 hours before the total weight was recorded. The labels were ashed in Trapelo low-temperature dry asher-505 to destroy the organic material and retain the volatile trace inorganics. The ashed labels were then acid digested with 1:1 nitric acid, filtered through S & S blue ribbon paper and diluted to 10 ml with demineralized water. The samples, standards, and blanks were all treated in the same above manner and analyzed on the atomic absorption spectrophotometer.

Air samples were analyzed by the atomic absorption technique (Thompson, 1970).

The soil and dust samples were also analyzed using the above method, except for a slight modification in pretreatment. All samples were digested with 1:1 HNO_3 instead of the suggested nitric-hydrochloric mixture.

Results are expressed as parts per million of lead content ($\mu g/g$). Where appropriate, expected normal values will be given.

Statistical Analyses

All statistical analyses were carried out utilizing the Mann–Whitney *U* test.

RESULTS

Air Samples

Table 1 depicts the mean traffic density and the mean concentrations of lead in the air at 2 and 5 ft. from the ground, at 12–15 ft. from one suburban and four city streets. The mean concentrations of airborne lead at the four city locations varied from 1.0–7.7 $\mu g/m^3$ at both heights. The range (95% confidence limits) was 0.7–9.9. The air in front of one apartment building on Street 3, a major entry point for two interstate highways where two study children lived, contained greater than 7 $\mu g/m^3$ of lead, more than twice the amount of lead found in the air at any of the other sites. The differences between the lead concentrations at 2 and 5 ft. are not significant ($P > 0.5$). The combined mean lead values for the two heights were 3.2 and 3.1 $\mu g/m^3$. The air at all four city sites was more leaded than the air at the suburban site, although the traffic was heavy at the latter site because of a detour. There was no correlation between traffic density and air lead levels.

TABLE I

AIR LEAD CONCENTRATION AT 2 FT. AND 5 FT. AND TRAFFIC DENSITY AT FOUR URBAN AND ONE SUBURBAN LOCATIONS

Site	Average No. cars/hr	No. Paired samples	Hours of sampling	Mean lead content $\mu g/m^3$			
				Distance from ground			
				2 ft.	5 ft.	Combined 2 ft. and 5 ft.	95% Confidence limits
Suburban street[a]	704	6	3–5 PM	1.0	1.1	1.04	0.7–1.3
City 1	259	6	8–10 AM	1.7	1.8	1.73	1.3–2.2
Housing project	40	5	8–10 AM	2.2	2.5	2.28	1.6–2.9
City 2	967	6	3–5 PM	3.2	3.0	3.06	2.4–3.7
City 3	1100	6	3–5 PM	7.7	7.1	7.30	3.8–9.9

[a] House on detour route at time of study.

TABLE 2

LEAD CONCENTRATIONS IN HARTFORD DIRT AND HOUSE DUST AND DIRT ON HANDS OF STUDY CHILDREN

Category	No. dwellings			No. samples			Lead conc. (μg/g)	95% Confidence Limits (μg Pb/g[a])
	Study	Control	Total	Study	Control	Total		
Outdoor dirt	11	6	17	94	29	123	1,200	700–1750
House dust	12	4	16	46	13	59	11,000	4,900–17,000
	No. children							
Hands[b]	10	6	16	16	6	22	2,400	650–4100

[a] For individual determinations.

[b] Mean sample weight was 11 mg which represents only a small fraction of the total amount of surface dirt present on hands.

Dirt, Dust, and Hand Dirt Samples

Table 2 shows the means and ranges of concentrations of lead in dirt in and around 11 houses of the ten study children and those of six controls. It is evident that the mean lead content of 1200 µg/g and range of lead concentrations (700–1750 µg/g) in the dirt are considerably above the average for the earth's crust (16 µg/g) (Swain, 1955). There were no significant differences between lead content of dirt surrounding study and control houses, so results were combined.

The mean level of lead in the house dust from 12 dwellings of ten study children, and houses of four of the six controls was 11,000. The differences between the lead content of the house dust of study children compared with controls was of borderline significance ($P = 0.05$), but because of small numbers, results are combined. The dust concentration of lead was ten times greater than that in dirt and this difference was highly significant ($P = < 0.001$). Two-thirds of the dust samples exceeded 1,000 µg/g lead, and several had more than 1% (10,000 µg/g). The highest concentrations of lead in indoor dust were found on windowsills, regardless of proximity to the street or whether the surfaces were painted. The content of lead in floor dust was next in order. The few specimens obtained from porches with painted railings were high in lead content.

The lead content in both the dust and dirt in the housing project which had two study and two control families was significantly lower than in and around nonproject dwellings ($P < 0.001$).

In Table 2, results are also depicted for 22 hand samples. Sixteen were from study children, and the remainder were from the six control children. The mean hand sample lead concentration was 2400 µg/g. The mean weight of hand dirt samples was 11 mg. This represents only a small fraction of the total amount of surface dirt present in the hands since much of it is trapped in skin folds and creases.

The difference in concentration of lead on the hands of study children compared with controls was not significant, so that results were combined in the table ($P > 0.05$); however, the hands of four project-dwelling children, two study children, and two controls, had significantly lower concentrations of lead than all of the others ($P < 0.05$ and > 0.01). The difference between lead content on the hand samples obtained when children were playing outdoors was not significantly different from those obtained when children were playing inside the dwelling ($P > 0.05$).

Observations of Mouthing Behavior

Nine of the ten study children exhibited some degree of excessive mouthing behavior during 3–6 hours of observation. Three were assigned a score of 4, meaning that hands or nonfood objects were in the mouth ≥ 50% of the time; only one had a score of 1. The mean score for the group was 3, indicating that most of the children had hands or nonfoods in the mouth frequently. The direct ingestion of paint, dirt, or dust was not observed. Based on the overall frequency with which mouthing behavior occurred and considering the wide variability of its occurrence within such a select population of children, a theoretical mouthing frequency of ten times a day was chosen as an extremely reasonable, if not conservative, estimate of the prevalence of hands or nonfood items being placed in the mouths of these children.

Results of Analyses of Water Samples

Only one of the 14 water samples contained any measurable lead and the quantity was well below the acceptable standard of 50 parts/billion.

Results of Testing of 18 Canned Food and Beverages and Four Nail P[illegible] Preparations

All but one of the foods sampled, but none of the nail polish samples contained measurable lead. The highest concentration in a canned food item was [illegible].0 µg/g, the lowest was < 0.01 µg/g; the mean lead content of 18 samples was [illegible] µg/g. The current estimate is that there is an average of 0.2 µg/g of lead in food[4] (thus, an intake of 1000 g of food per day could provide 200 µg of lead).

DISCUSSION

Lead in Air

The annual average "hi-vol" lead air quality data for the City of Hartford from 1966–1972 are summarized in Table, 3,[5] the elevation above ground of each of these sites is 30 ft. or greater, and the range of lead concentrations was [illegible]2.0 µg/m³. The lead content of the 2-hour peak traffic ground level air samples taken during the present study cannot be compared quantitatively to the hi-vol data since the sampling methods were different. However, urban children are typically exposed to ground level daytime lead concentrations, while playing outdoors at less than 20 ft. from the street, and it appears that these children may be subjected to much higher lead levels than would be indicated by the hi-vol sampling > 30 ft. above the ground. Similar data have been reported by [illegible] (1973), although those studies in Brattleboro, Vermont showed a significant dif-

TABLE 3
SUMMARY OF THE HARTFORD LEAD AIR QUALITY DATA, ANNUAL AVERAGES

Site location	1966	1967	1968	1969	1970	1971	1972
Public Library[a]	—	—	—	—	1.6	1.9	2.0
Hartford Health Department[b]	—	—	—	1.6	—	—	1.6
State Health Department[c]	1.4	—	0.6	1.0	—	—	—

[a] µg/m³.
[b] Elevation of site above ground is 30′.
[c] Elevation of site above ground is 50′.

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[4] Evaluation of certain food additives and the contaminants, mercury, lead, and [illegible]. Sixteenth Report of the Joint FAO/WHO Expert Committee on Food additives. WHO Technical Report Series No. 505. FAO Nutrition Meeting Report Series No. 51. Geneva, Switzerland, [illegible]

[5] Determined by the Air Compliance Unit of The Connecticut State Department of Environmental Protection.

ference in lead content of air below and above 4 ft from the ground level.

The lack of correlation between lead concentration in air and traffic density at the sampling site in the present study may be accounted for by the following reasons:

(1) Adjacent street traffic density might affect the ambient air lead levels at many sites since the suspended lead particles are typically sub-micron in size and, therefore, would be carried long distances before deposition. It would have been more accurate to consider a sample site located at the center of one square mile/day. In one study in Los Angeles, a correlation of 0.964 was reported between airborne pollutants and traffic density. (Gordon, 1973)

(2) Local meterological conditions were neglected.

(3) The effects of street width, presence of tall buildings, and movement of traffic were not evaluated.

The findings of excessive lead content in dirt near heavily trafficked streets in the present study compared well with those of Darrow (1973) and the report of Hunt (1971) while the house dust data of the present study confirm the findings of Sayre *et al.*, 1974. Although TerHarr's (1974) findings implicate paint as major source of lead in soil less than 2 ft. from the house, it cannot be the principal source of lead near roadways where children play.

The higher lead concentrations in dust indoors than in outdoor dirt may be accounted for by a number of factors including different mechanisms and rates of removal such as wind, rain, and fallout outdoors; housekeeping practices; and the presence of old pulverized leaded paint indoors. The fallout of lead from air, which in the opinion of the authors originated largely from gasoline emissions, is a significant contributor to the lead present in the dirt and dust found in urban streets, parks, and homes, since high lead concentrations were found in dirt and dust well removed from painted surfaces.

Although the absolute relationship between ambient air lead and that in dirt and dust must be further defined before air quality standards for lead can be established, it is clear from the data herewith presented that the levels of lead found in the air near the ground and in the dirt and dust are far too high.

The maximum daily permissible lead intake (MDPI) for a preschool child from all sources has been estimated to be 90–500 μg (King, 1971), assuming that absorption as in the adult is approximately 10%. The potential daily lead intake from different categories of environmental exposure is depicted in Table 4. In certain instances, the MPDI could be exceeded if there were excessive contamination by lead of food and water. This does not appear to be the situation in the present study group. Clearly, leaded paint is potentially a highly serious problem because of the high lead concentration; consequently, the repetitive ingestion of even fractions of grams of leaded paint could raise a child's daily lead intake to more than 100 times the highest MDPI and could result in high levels with severe symptoms. Evidence has been presented herewith that the ingestion of 0.25g of Hartford street dirt (1200 μg/g) or house dust (11,000 μg/g) might result in an extra daily intake of 300 or 2750 μg of lead, respectively. In addition, if a small child playing in such a contaminated environment puts his hands or fingers in his mouth ten times each day he could theoretically ingest 240 μg of lead, thus approaching the MDPI from this source alone. Consequently, it is probably that a

TABLE 4

ESTIMATED DAILY LEAD INTAKE FOR A 2 YEAR OLD CHILD BY EXPOSURE ROUTE[a]

Category	Potential daily Lead intake (μg)
Air (respired)[b]	6–60
Ingested	
Paint	50,000[c]
Dust	2,750[d]
Dirt	300[e]
Dirt on hands	240[f]
Food and water	130–170

[a] MDPI, 90–500 μg.

[b] Assumed intake 6m^3 air with 1–10 $\mu g/m^3$ lead.

[c] 0.25 g paint chip with 20% lead.

[d] 25 mg of dust with 11,000 $\mu g/g$ lead.

[e] 25 mg of dirt with 1,200 $\mu g/g$ lead.

[f] 2400 $\mu g/g$ lead in 10 mg dirt ingested ten times.

young child who puts his hands in his mouth frequently during the day in such an environment could accumulate an increased body burden of lead. The implications of these findings are far-reaching, because society has to be accountable for allowing such a significant health hazard to exist that may well affect the life and health of millions of urban children. While the investigators believe that lead emitted from automobiles contributes significantly to contamination of air, dirt, and dust, the environmental impact of removing lead from gasoline is not fully understood. Accordingly, an alternative might be mass transit utilizing a different form of energy.

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The Arnhem Lead Study[1]

I. Lead Uptake by 1- to 3-Year-Old Children Living in the Vicinity of a Secondary Lead Smelter in Arnhem, The Netherlands

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An epidemiological study was carried out in the vicinity of a secondary lead smelter in Arnhem, The Netherlands. Blood lead levels of 1- to 3-year-old children were determined, and proved to be slightly elevated. In addition, a number of environmental parameters were determined, including lead in air, dustfall, soil, streetdust, several types of housedust, and drinking water. Statistical analysis showed that lead in garden soil, lead in dustfall outdoors, and lead in dustfall indoors were the most important parameters for the explanation of variance of blood lead levels in the area under study.

INTRODUCTION

Blood lead levels (PbB's) of children living in the vicinity of lead smelters tend to be higher than those of adults. This suggests that there is a higher uptake and/or absorption of lead in children than there is in adults, in such environments.

An important practical question concerns the routes of uptake. Food, ambient air, soil, streetdirt, housedust, drinking water, and paint have all been described as possible routes of lead uptake from the environment.

In the vicinity of lead smelters, special emphasis has been laid on ambient air, soil, and housedust (Yankel *et al.*, 1977; Landrigan *et al.*, 1975). In Arnhem, a city of 150,000 inhabitants in the eastern part of The Netherlands, a secondary lead smelter has been in operation for the past 30 years. A survey of seven hundred 1- to 7-year-old children living in the vicinity of the smelter showed that PbB's increased between the ages of 1 and 4, and that the Biological Quality Guide, as accepted by the Commission of the European Communities (CEC), was slightly exceeded at distances of less than 600 m from the source.

The CEC Biological Quality Guide states that in the general population a PbB of 400 μg/liter should not be exceeded, that 98% of PbB's should be below 350 μg/liter, 90% below 300 μg/liter, and 50% below 200 μg/liter. The lead emission of the smelter was in the recent past probably as high as 500 kg Pb per day, but has now dropped to 50 kg per day. It was felt, however, that the presence of large amounts of lead, deposited over the years in the immediate surroundings of the smelter, still could cause problems for several years in the future, as lead tends to be very persistent in soils. Therefore, the Agricultural University in Wageningen was requested by the Municipal Council of Arnhem to investigate along which main routes the children were exposed to lead. The purpose was to find out how the lead uptake by small children could be diminished in the most effective way.

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An epidemiological model was used, with PbB as a dependent variable, and several environmental, behavioral, and other parameters as independent variables.

MATERIALS AND METHODS

Blood lead levels were determined in a sample of ninety-five 1- to 3-year-old children living within 1000 m from the smelter, or about half of all children in this age group living in the area. The one year olds were slightly under-represented in the group that was studied. All blood samples were taken by venous puncture in 1 week in May 1978. Analyses were performed by the Coronel Laboratory in Amsterdam. The methods have been described by Castilho and Herber (1977).

During the weeks before the blood samples were taken, a comprehensive environmental study was carried out. The variables that were determined are listed in Table I. In addition, a brief history of each child, including age, sex, length of residence, education and occupation of parents, mouthing behavior, length of time spent indoors and outdoors, etc., was taken. Furthermore, a subgroup of 39 children was observed for 1 or 2 days during normal play. These children were mostly one and two year olds. It was assumed that in the small and socially relatively homogeneous study area, no systematic differences in food supply and diets would occur.

TABLE I

ENVIRONMENTAL PARAMETERS AND METHODS: ARNHEM LEAD STUDY, 1978[a]

Parameter	Method
1. Lead in ambient air	High-volume samples: 24-hr measurements at 6 sites, continuously for 2 months
2. Lead in dustfall	Standard deposit gauges: 7-day measurements at 22 sites, semicontinuously for 3 months
3. Lead in soil	Sampling in gardens of study population; analysis of layers from 0 to 5 cm and 5 to 20 cm
4. Lead in streetdust	Samples at 31 sites, analysis of fraction < 0.3 mm
5. Lead in indoor air	Low-volume samples; 1-month measurements in homes of study population, continuously for 2 months
6. Lead in dustfall indoors	Greased glass plates of 30 × 40 cm; 1-month measurements in homes of study population, continuously for 3 months
7. Lead in floordust	Vacuum cleaner with special filter holder; 5 samples, collected on 3 different occasions, with intervals of approximately 1 month, in homes of study population
8. Easily available lead indoors	Wet tissues, 1 sample in homes of study population
9. Lead in tapwater	Proportional samples, during 1 week in homes of study population
10. Dustiness of homes	Visual estimation, on a simple scale ranging from 1 (clean) to 3 (dusty); 6 observations in homes of study population

[a] All lead analyses were performed by atomic absorption spectrophotometry, except part of the tapwater analysis, which was performed by anodic stripping voltametry. Lead in tapwater analyzed by the National Institute of Drinking Water Supply in Leidscherdam. Soil and streetdust analyzed by the Laboratory of Soil and Plant Research in Oosterbeek.

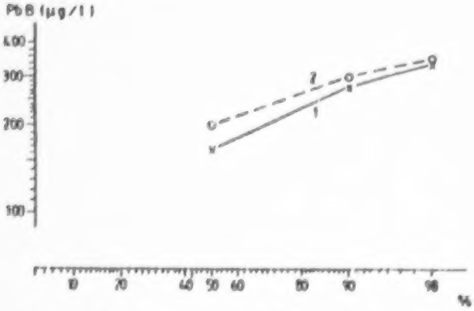

FIG. 1. Cumulative distribution of blood lead levels in 1- to 3-year-old children (1), as compared to the CEC Biological Quality Guide (2). Arnhem Lead Study, 1978.

No use of self-grown vegetables was recorded in the study area. Lead contents of peeling paint were determined in a few houses where exceptionally high lead concentrations in housedust were found. Other possible sources of lead (pottery, colored print, toothpaste, etc.) were not investigated.

Simple and multiple regression analyses were used to determine which (combination of) independent variables could best explain the variance of the dependent variable.

RESULTS

The geometric mean of blood lead levels was 161 µg/liter. The distribution of PbB's was slightly below the CEC standard (see Fig. 1).

The results of the environmental measurements are summarized in Table 2. Results of the observations of dustiness and child behavior will not be presented as such, but only in relation to PbB's.

A high lead content (up to 40% by weight) in peeling paint was found in a few old houses (built around 1900), where extremely high lead contents in housedust had

TABLE 2

ENVIRONMENTAL POLLUTANT CONCENTRATIONS IN ARNHEM: ARNHEM LEAD STUDY, 1978

Parameter	Geometric mean	Arithmetic mean	Range
1a. Lead in ambient air ($\mu g/m^3$, 2 months' average)	0.41	0.42	0.28–0.52
1b. TSP ($\mu g/m^3$, 2 months' average)	64.0	64.5	53.7–73.0
2. Lead in dustfall ($\mu g/m^2$/day)	467	551	108–2210
3. Lead in soil (mg/kg, 0–5 cm)	240	322	21–1126
4. Lead in streetdust (mg/kg, < 0.3 mm)	690	859	77–2667
5. Lead in indoor air ($\mu g/m^3$, 2 months' average)	0.26	0.27	0.13–0.74
6a. Lead in dustfall indoors ($\mu g/m^2$/day)	7.34	9.32	1.36–42.35
6b. Lead content of deposited dust (mg/kg)	1007	1144	457–8097
7a. Lead in fine floordust (mg/kg)	957	1054	463–4741
7b. Lead in coarse floordust (mg/kg)	282	370	117–5250
8. Easily available lead indoors	85% of samples < 20 µg Pb/tissue		
9. Lead in tapwater (µg/liter)		5.0	<0.5–90.0

TABLE 3

Simple Correlations between log PbB and 22 Independent Variables: Arnhem Lead Study, 1978

No.	Description	r log PbB	n	Significance
1	Lead content of fine floordust (mg/kg), mean value of 5 samples	−0.045	95	n.s.
2	Lead content of coarse floordust (mg/kg), mean value of 5 samples	−0.028	95	n.s.
3	Amount of floordust sampled (mg/m^2), mean value of 4 samples	0.204	95	$P < 0.05$
4	Amount of lead sampled on floors (μg/m^2), mean value of 4 samples	0.089	95	n.s.
5	Lead content of deposited dust indoors (mg/kg), mean value of 3 samples	−0.005	93	n.s.
6	Amount of dust deposited indoors (mg/m^2/day), 3 months' average	0.086	93	n.s.
7	Amount of lead deposited indoors (μg/m^2/day), 3 months' average	0.178	93	$P < 0.05$
8	Lead in indoor air (μg/m^3), 2 months' average	0.090	89	n.s.
9	Lead in ambient air (μg/m^3), 2 months' average	0.031	95	n.s.
10	Amount of lead deposited outdoors (μg/m^2/day), 2 months' average	0.059	95	n.s.
11	Lead in soil toplayer, 0–5 cm (mg/kg)	0.177	62	n.s.
12	Lead in streetdust, fraction smaller than 0.3 mm (mg/kg)	0.085	95	n.s.
13	Dustiness of the home, scale ranging from 6 (clean) to 18 (dusty), based on 6 independent observations	0.252	95	$P < 0.01$
14	Distance from the smelter, intervals of 100 m	−0.014	95	n.s.
15	Educational level of parents: 1 = low; 2 = high	−0.105	95	n.s.
16	Age of the child in months	0.230	94	$P < 0.05$
17	Average number of times a hand is put into the mouth per 10 min	0.262	33	n.s.
18	Average number of times a dirty object is put into the mouth per 10 min	0.325	33	$P < 0.05$
19	Average number of times an object (dirty or clean) is put into the mouth per 10 min	0.152	33	n.s.
20	Average number of times on object (dirty or clean) or a hand is put into the mouth per 10 min	0.252	33	n.s.
21	Daily average number of times the child's hands were cleaned	0.044	33	n.s.
22	Average number of hours per day the child did not sleep	0.210	33	n.s.

been found during the survey. The children living in these houses were excluded from further analysis.

The results of the housedust survey will be reviewed separately in part II of this study.

Simple correlation coefficients were calculated for the relationship between log PbB and 22 different independent variables. The results are shown in Table 3. Significant correlations were found between log PbB and the quantity of sampled floordust, quantity of deposited lead indoors, observational score of dustiness, age of the child, and average number of times a dirty object is put in the mouth.

Interpretation of these simple correlation coefficients is difficult, as many of the independent variables will be interrelated. For this reason, multiple regression techniques were used to assess the relationship between PbB and combinations of independent variables. The children were divided into some more or less homogeneous subgroups before the analysis was carried out. The results are shown in Table 4.

Most important were the following subgroups:

A. children living in houses with a garden ($n = 53$),

B. children living in houses without a garden ($n = 27$).

In subgroup A, the combination of soil lead level and educational level of the parents explained 23% of the variance of log PbB ($R^2 = 0.23$, $P < 0.01$). In subgroup B, the amount of deposited lead indoors explained 26% of the variance of log PbB ($R^2 = 0.26$, $P < 0.01$).

Another significant correlation was the one between log PbB and lead deposition outdoors, for children who often play outdoors ($R^2 = 0.21$, $P < 0.05$, $n = 24$). Multiple regression analysis was also carried out on a subgroup of 32 of the children whose behavior had been observed.

The independent variables 16–22 (cf. Table 3) were used. A significant correlation was found between log PbB and the combination of the average number of times a dirty object was put into the mouth per 10 min, and the number of hours per day the child was awake ($R^2 = 0.22$, $n = 32$, $P < 0.05$). It was not possible to find any significant relationship between log PbB and *combinations* of environmental and behavioral variables.

DISCUSSION

Compared with the situation around other lead smelters abroad, the surroundings of the Arnhem lead smelter were not extremely polluted with lead. Much higher environmental concentrations have been reported around lead smelters in Canada and the United States (Roberts *et al.*, 1974; Landrigan *et al.*, 1975; Yankel *et al.*, 1977). Even so, blood lead levels in Arnhem children were correlated with lead in topsoil, lead deposition indoors, and lead deposition outdoors. This is in line with the literature, which indicates soil, air, and housedust as the most important potential routes along which small children take up lead from their environment.

At the present time, an intervention is planned to diminish the lead content of garden soil and streetdirt in the area under study. The effects of this intervention on PbB levels of small children will be evaluated subsequently.

TABLE 4

RESULTS OF MULTIPLE REGRESSION ANALYSIS BETWEEN log PbB AND A NUMBER OF INDEPENDENT VARIABLES: ARNHEM LEAD STUDY, 1978

Group characteristics	R^{2a}	n	P	Variables[b]	Equation
Playing outdoors often	0.21	24	<0.05	10	log PbB = 1.945 + 0.0008 var 10
Playing indoors often		23			no significant relationships
With a garden	0.23	53	<0.01	11,15	log PbB = 2.356 + 0.0003 var 11 −0.19 var 15
high-education parents		8			no significant relationships
low-education parents	0.20	45	<0.01	11	log PbB = 2.153 + 0.0003 var 11
Without a garden	0.26	27	<0.01	7	log PbB = 2.055 + 0.136 var 7
high-education parents		2			no significant relationships
low-education parents	0.32	25	<0.01	7	log PbB = 2.035 + 0.143 var 7
Behavior observed	0.22	32	<0.05	18,22	log PbB = 1.706 + 0.044 var 18 + 0.32 var 22

[a] The R^2 value is the fraction of variance in log PbB explained by the given variable(s).

[b] For explanation of variable numbers see Table 3. When only one variable is mentioned, this means that it is the only variable out of several that conibutes significantly to the explanation of the variance of log PbB, for the given subgroup. As can be seen from the results, a combination of two variables ontributes significantly to the explanation of the variance of log PbB only in two cases.

Quantitative relationships between environmental lead and PbB, based on epidemiological studies, are few at this moment. Barltrop (1975) found a significant correlation between PbB in 2-year-old children and soil lead, in a situation where soil lead ranged from less than 1000 mg/kg to more than 10,000 mg/kg. In the soil lead range from 420 to 3390 mg/kg, PbB's were shown to increase with 5 μg/liter for every 500 mg/kg increase in soil lead. In the range from 3390 to 13,969 mg/kg, PbB's increased with 2.5 μg/liter for every 500 mg/kg increase in soil lead.

Yankel *et al.* (1977) found significant correlations between PbB in 1- to 9-year-old children and lead in ambient air and topsoil, in the vicinity of a lead smelter in Kellogg, Idaho. Soil lead concentrations ranged from 50 to 14,600 mg/kg. PbB's were shown to increase with 3 μg/liter for every 500 mg/kg increase in soil lead, in the range from 500 to 5000 mg/kg.

From the equations in Table 4, a quantitative relationship between soil lead and PbB can be assessed for the situation in Arnhem. A soil lead increase from 100 to 600 mg/kg—the range in whch most soil lead levels in Arnhem were found—produces here an increase in PbB's of 63 μg/liter. Clearly, the regression line in this study is much steeper than the ones cited from the literature. An explanation could be that the total lead uptake in Arnhem, as reflected in the PbB-levels, was smaller than the total lead uptake in the groups studied by Barltrop and Yankel *et al.* As has been shown by Angle *et al* (1979), the increase of PbB per unit increase of exposure is greater at low levels of total lead uptake than at high levels of total lead uptake.

The amount of lead deposited indoors represents the housedust fraction which is most readily available to small children. This fraction settles on every surface in the room, and a child can swallow it inadvertently when putting its fingers or other objects into its mouth. Housedust, sampled by vacuum cleaner, is presumably much harder to reach for small children, as it is pulled out of carpets, rugs, etc. by force.

The conclusion would be that it is better to sample for settlable dust indoors than for floordust if one wants to characterize the indoor dust pollution fraction, which has the most biological significance. The air and tissue samples did not yield important epidemiological information in this study. This, however, could well be due to the relatively low overall pollution levels in Arnhem. In a different situation, the collection of air and tissue samples would probably prove to be more useful.

CONCLUDING REMARK

The fact that significant relations between blood lead and environmental lead could be demonstrated, although in general the environmental lead concentrations in Arnhem were not exceptionally high, suggests that probably in several inner-city areas with heavy traffic, lead uptake by small children leads to the same or even higher blood lead levels than those found in Arnhem. To date, however, no data are available on blood lead levels of small children in Dutch inner-city areas.

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The Arnhem Lead Study[1]

II. Indoor Pollution, and Indoor/Outdoor Relationships

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As part of an epidemiological study, an extensive investigation was carried out on the pollution of several types of housedust with lead. Samples were taken of ambient air indoors, lead deposition indoors, floordust, and dust deposited on windowsills, etc. In addition, the dustiness of houses was estimated visually. A number of outdoor parameters were determined, which made a detailed indoor/outdoor comparison possible. In general, indoor pollution levels were lower than the corresponding outdoor levels. Statistical analysis showed that in Arnhem, lead is carried into the houses in the form of lead particles that adhere to shoes, clothes, etc. Most of it originates from gardens and possibly streetdirt.

INTRODUCTION

In recent years, much attention has been given to housedust as a potential source of lead uptake by small children. Little agreement exists, however, on the relative importance of different types of housedust, and as to how housedust actually gets polluted with lead or other elements with a predominantly outdoor origin. Samples have been taken of floordust, with a special vacuum cleaner (Solomon and Hartford, 1976) or with the vacuum cleaner present in the house (Yankel *et al.*, 1977), of lead deposition indoors (Day *et al.*, 1977), of indoor air (Johnson *et al.*, 1978), of lead deposited on windowsills, etc. (Sayre *et al.*, 1974).

Seldom have several types of housedust been sampled at the same time (the epidemiologic study described in Johnson *et al.*, 1978, is an exception). Also, the interrelationships between several types of housedust and the relative availability of the different types of housedust for uptake by small children have never been investigated in detail. Studies of indoor/outdoor relationships do exist, but most of them consider only gases or suspended particulate matter in air (cf. Alzona *et al.*, 1976; Sterling and Kobayashi, 1977).

In the city of Arnhem, located in the eastern part of the Netherlands, an epidemiological study was carried out in 1978 to determine the major environmental pathways of lead uptake by small children living in the vicinity of a secondary lead smelter. Part of the study consisted of an extensive investigation of the lead pollution of several types of housedust. Also, a number of outdoor parameters was determined, allowing a detailed indoor/outdoor comparison. The epidemiological results are presented in part I of this study (Brunekreef *et al.*, 1981). In this paper, attention will focus on the methods and results of the housedust investigation, and on the indoor/outdoor relationships. Over 100 houses were investigated, situated in the nearest residential area, at distances of 450–1000 m north of the

[1] [illegible] by a grant from the Municipal Council in Arnhem.

smelter (cf. Fig. 1). The parameters investigated were: suspended particulates in indoor air, dustfall indoors, floordust, lead on windowsills and other flat surfaces, dustiness in general, and, in a few cases, lead in peeling paint.

MATERIALS AND METHODS

Suspended particulates in indoor air were sampled with a small aquarium pump (Rena 101) adapted for air-sampling purposes. The pump was connected to a Schleicher and Schüll FP 050/02 filter holder with glass-fiber filters (Whatman GF/C) of 47-mm diameter. In each house, two continuous samples of 28 days were collected. Wherever possible, the equipment was placed in the living room at a height of approximately 1.5 m. Filters were weighed before and after sampling, to establish the weight of the collected samples. The flow rate was 1.0–1.5 litres per minute, resulting in a linear velocity of 1.5–2.0 cm/sec. Thus, according to Davies' criteria, particles smaller than 3–4 μm in diameter will be sampled efficiently (Davies, 1968). The flow rate was checked three times per sampling period with a rotameter. In 19 cases, sampled volumes were measured with dry gasmeters.

Dustfall indoors was sampled with 30 × 40-cm glass plates (cf. Day *et al.*, 1977). The plates were greased by pouring a mixture of paraffin (viscosity greater than 110 cP at 20°C) and petroleum ether 40/60 on them and allowing the petroleum ether to evaporate. The plates were placed in the living rooms of the houses, at a height of approximately 2 m, usually on cupboards. Three continuous samples of 28 days were taken. After sampling, the paraffin plus collected dust was removed by rinsing the plates with petroleum ether 40/60. The suspension was centrifuged at 4000 rpm for 5–10 minutes. After decanting the paraffin/petroleum ether mixture, the dust was resuspended in petroleum ether and collected on a preweighed cellulose filter (Schleicher and Schüll 602 h, diameter 55 m). The centrifugation–resuspension step was necessary to avoid irreversible paraffin uptake by the cellulose filters.

Floordust was sampled with an ordinary vacuum cleaner (FAM IS 1000, 650 watts), with an aluminum filter holder (cf. Solomon and Hartford, 1976). Whatman GF/C glass-fiber filters of 150-mm diameter were used. Samples were collected on

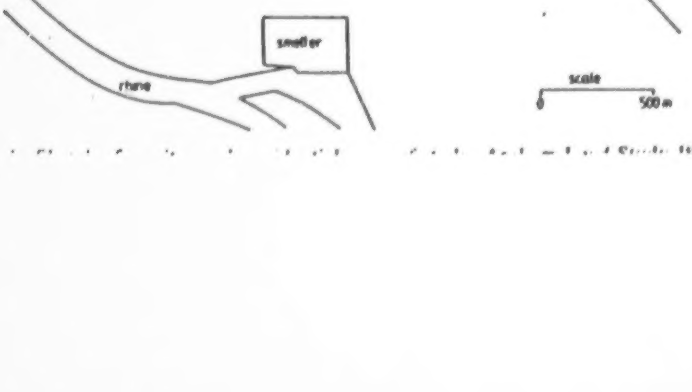

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TABLE 1
MEAN LEVELS OF INDOOR LEAD AND DUST POLLUTION: ARNHEM LEAD STUDY, 1978

Parameter	Geometric mean	Arithmetic mean	Range	n[a]
Suspended particulate matter				
dust concentration (μg/m^3)	120	140	20–570	101
lead concentration (μg/m^3)	0.26	0.27	0.13–0.74	101
dust lead content (mg/kg)	2290	2670	400–8200	106
Dustfall				
dust deposition (mg/m^2/day)	7.7	15.0	1.4–63.9	105
lead deposition (μg/m^2·day)	7.34	9.30	1.36–42.35	105
dust lead content (mg/kg)	1007	1144	457–8097	105
floor dust				
amount of dust (mg/m^2)	255	356	41–2320	107
amount of lead (μg/m^2)	123	166	18–886	101
dust lead content (mg/kg)				
in "fine" floor dust	957	1054	463–4741	107
in "coarse" floor dust	282	370	117–5250	101

[a] n, number of houses.

three occasions in each house, with intervals of approximately 28 days. During the first sampling period, only one sample per house was collected. In the second and third, two samples were collected, using a wooden frame to obtain a standard sampling surface of 1 m^2. After weighing total dust, the "coarse" floordust fraction, resting loose on the filter, was removed and analyzed separately. The filter with the remaining "fine" floordust fraction was weighed again and subsequently prepared for analysis.

Lead on windowsills and other flat surfaces was sampled with Kleenex "professional use" tissues (cf. Sayre *et al.*, 1974). The tissues were moistened with a solution of ethanol (1:5) and benzalkonium chloride (1:750) in water. The method

TABLE 2
MEAN LEVELS OF OUTDOOR LEAD AND DUST POLLUTION: ARNHEM LEAD STUDY, 1978

Parameter	Geometric mean	Arithmetic mean	Range
Suspended particles			
dust concentration (μg/m^3)	64.0	64.5	53.7–73.3
lead concentration (μg/m^3) (high-volume samplers, 24-hr samples, 2 months' average)	0.41	0.42	0.28–0.52
Lead in dustfall			
(μg/m^2/day) (deposit gauges, weekly samples, 2 months' average)	467	508	208–2210
Lead in soil			
(mg/kg, 0–5 cm)	240	322	21–1126
Lead in streetdust			
(mg/kg, < 0.3 mm)	690	859	77–2667

TABLE 3a

SELECTED CORRELATION COEFFICIENTS BETWEEN LEAD CONCENTRATIONS OF INDOOR AND OUTDOOR PARAMETERS: ARNHEM LEAD STUDY, 1978

Parameters	r	n	
Soil – fine floordust	0.40	71	
Soil – coarse floordust	0.17	71	n.s.[a]
Soil – dustfall indoors	0.35	70	
Soil – suspended particles indoors	0.53	70	
Fine – coarse floordust	0.71	101	
Fine floordust – dustfall indoors	0.76	105	
Fine floordust – suspended particles indoors	0.49	106	
Coarse floordust – dustfall indoors	0.39	101	
Coarse floordust – suspended particles indoors	0.35	101	
Dustfall indoors – suspended particles indoors	0.41	105	

[a] n.s., not significant ($P \geq 0.05$).

tries to simulate the moist hand of a child. Dust was collected by wiping surfaces that are accessible to children, such as windowsills, low tables, etc.

Lead in samples was analyzed by atomic absorption spectrophotometry. The samples were digested in 10 N HCl according to a standard procedure (Dutch Institute for Standardization, 1977) and diluted before analysis. The recovery of this method is estimated to be at least 95%, based on experiments conducted in our own laboratory.

The general dustiness of each house was estimated three times by two independent observers, during dust-sampling visits. A simple scale of 1 (clean) to 3 (dusty) was used (cf. Yankel *et al.*, 1977). By adding the individual values, the score for each house was calculated.

RESULTS

Mean levels of indoor lead and dust pollution are shown in Table 1. For comparison, the results of a number of outdoor measurements are summarized in Table 2.

Correlation coefficients were computed between a number of parameters. The

TABLE 3b

SELECTED CORRELATION COEFFICIENTS BETWEEN AMOUNTS OF SAMPLED LEAD IN INDOOR AND OUTDOOR PARAMETERS: ARNHEM LEAD STUDY, 1978

Parameters	r	n	
Total floordust – dustfall indoors	0.45	105	
Total floordust – suspended particles indoors	0.23	101	
Dustfall indoors – suspended particles indoors	0.26	99	
Dustfall indoors – dustfall outdoors	0.03	105	n.s.[a]
Suspended particles indoors – outdoors	0.08	101	n.s.[a]
Dustfall outdoors – suspended particles outdoors	0.80	30	
Dustfall outdoors – soil	0.10	38	n.s.[a]

[a] n.s., not significant ($P \geq 0.05$).

TABLE 3c

SELECTED CORRELATION COEFFICIENTS BETWEEN AMOUNTS OF SAMPLED DUST IN INDOOR AND OUTDOOR PARAMETERS: ARNHEM LEAD STUDY, 1978

Parameters	*r*	*n*	
Total floordust – dustfall indoors	0.46	105	
Total floordust – suspended particles indoors	0.19	101	
Dustfall indoors – suspended particles indoors	0.16	99	n.s.[a]
Dustfall outdoors – suspended particles outdoors	0.39	30	

[a] n.s., not significant ($P \geq 0.05$).

results are given in Tables 3a (lead concentrations of samples), 3b (amounts of lead in samples), and 3c (amounts of dust in samples). Table 4 shows the correlation coefficient between the lead concentration in indoor air (μg Pb/m^3) and the concentration of suspended particulate matter in indoor air (μg/m^3). Also, the correlation coefficient between the lead content of suspended particulates (mg/kg) and the concentration of suspended particulate matter in indoor air (μg/m^3) is shown.

Sixty houses were selected in which all measurements were carried out in the living rooms of the houses. Included were 34 ground-floor houses and 26 second- and third-floor houses. The differences of pollution levels between the ground-floor houses and the second- and third-floor houses are given in Table 5.

DISCUSSION

Indoor lead pollution levels were lower than those found in studies around other lead smelters (Roberts *et al.*, 1974; Landrigan *et al.*, 1975; Yankel *et al.*, 1977). This is probably due to the fact that the Arnhem lead smelter is relatively small, and that there are no houses immediately adjacent to the source.

Lead concentration levels in indoor air in Arnhem were lower than those in outdoor air. The concentration of total suspended particulates was higher in indoor air than in outdoor air. This suggests the presence of indoor sources of airborne particulate matter (cf. also Table 4) like tobacco smoke, home repair, house cleaning, decay of furnishings, etc. (Sterling and Kobayashi, 1977).

The question remains how lead, present in suspended particulate matter indoors, enters the house. Alzona *et al.* (1976) suggest as a rule of thumb a ratio of 0.3 between indoor and outdoor concentrations of elements of outdoor origin in air, when resuspension of material that enters the room other than by airborne

TABLE 4

CORRELATIONS BETWEEN LEAD AND DUST IN INDOOR AIR: ARNHEM LEAD STUDY, 1978

Parameters	*r*	*n*	
Suspended particles (μg/m^3) – lead content of suspended particles (mg/kg)	−0.67	101	
Suspended particles (μg/m^3) – amount of lead (μg/m^3)	0.06	101	n.s.[a]

[a] n.s., not significant ($P \geq 0.05$).

TABLE 5

DIFFERENCES OF POLLUTION LEVELS BETWEEN GROUND-FLOOR HOUSES AND SECOND- AND THIRD-FLOOR HOUSES: ARNHEM LEAD STUDY, 1978

	Lead concentration of dust (mg/kg)			Amount of lead sampled (μg/units (s))[a]			Amount of dust sampled (mg/unit(s))[a]		
Parameter	1st floor	2nd and 3rd floors	p	1st floor	2nd and 3rd floors	p	1st floor	2nd and 3rd floors	p
Suspended particles	2050	2450	<0.005	0.270	0.245	n.s.[b]	0.173	0.113	<0.005
Deposition indoors	955	1240	n.s.[b]	10.4	6.5	<0.01	11.2	6.6	<0.005
Fine floordust	1012	814	n.s.[b]	197	116	<0.05	397	249	<0.005
Coarse floordust	258	296	n.s.[b]						

[a] For units see text.

[b] n.s., not significant ($P \geq 0.05$), t statistics evaluation, two-sample test.

means is excluded. The ratio found in this study is higher (0.62), suggesting a more important role of resuspension. The statistical associations between indoor parameters generally reach significance. However, the only significant indoor/outdoor relationships are those between soil lead content and lead contents of most indoor parameters (cf. Table 3).

It is possible that some of the other outdoor parameters show no significant correlation with indoor parameters because their range is small (outdoor air, deposition outdoors) and/or because the values of these outdoor parameters for each individual house have been estimated by interpolation (outdoor air, deposition outdoors, streetdust). In the case of streetdust, high and low values were distributed at random throughout the area under study. Therefore, it can hardly be expected that interpolation of the streetdust data yields reliable results. Still, the data suggest that most of the lead, present in housedust, is carried into the houses in the form of dust particles, adhering to shoes, etc. This finding can be supported by comparing the amounts of lead sampled by vacuum cleaning and with the glass plates (cf. Table 1). On the average, 166 μg Pb/m^2 was sampled from the floors, compared to a settling rate of 9.3 μg Pb/m^2 per day. The frequency of vacuum cleaning by the tenants is known to be once every 1 or 2 days. This suggests that much more lead is removed by vacuum cleaning than can be supplied by deposition.

The exact settling/removal ratio could not be determined, as no information was available on the eventual difference in sampling efficiency between the experimental sampler and the domestic vacuum cleaners. The difference between the pollution levels of ground-floor houses and second- and third-floor houses also supports the conclusion that most lead was carried into the house on shoes, etc. One or more staircases have to be climbed before the second- and third-floor houses are reached, and some of the adhering particles will be lost before the houses are entered. In these houses, no direct access to gardens is possible (Table 5).

Some of the remaining unknowns are: lead content in relation to size distribution of the different forms of sampled dust, and the mechanisms of dust resuspension indoors.

Floordust seems to be the intermediate pool of lead particles, entering the house from gardens and streets.

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slices were added to tubes containing [illegible] agents after incubation for 30 minutes and the other agents were added 10 minutes later. Incubations were terminated by placing the tubes in boiling water for [illegible] minutes. The samples were then centrifuged at low speed and duplicate 50-µl portions of each supernatant were analyzed for cyclic AMP as described by B. L. Brown, J. D. M. Albano, R. P. Ekins, and A. M. Sgherzi [*Biochem. J.* 121, 561 (1971)]. The protein content of the samples was determined by the method of O. H. Lowry, N. J. Rosebrough, A. L. Farr, and R. J. Randall [*J. Biol. Chem.* 193, 265 (1951)]. The cyclic AMP content was proportional to the size of the portion tested, and known amounts of authentic cyclic AMP added as an internal standard were quantitatively recovered. Cyclic AMP content measured by this method was the same as that found by homogenizing the tissue in a mixture of ethanol (98 percent) and HCl (0.2M), centrifuging, evaporating the supernatant to dryness, and redissolving the residue for determination of cyclic AMP as described previously (*11*). In experiments where the cyclic AMP content of the slices and the incubation medium were determined separately, virtually all the cyclic AMP was found in the slices. The data are expressed as picomoles of cyclic AMP per milligram of protein. Each point represents the mean ± the standard error of the mean (S.E.M.) for six or more replicate samples.

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11 August 1974 ■

Lead Contamination around Secondary Smelters: Estimation of Dispersal and Accumulation by Humans

Abstract. *A high rate of lead fallout around two secondary lead smelters originated mainly from episodal large-particulate emissions from low-level fugitive sources rather than from stack fumes. The lead content of dustfall, and consequently of soil, vegetation, and outdoor dust, decreased exponentially with distance from the two smelters. Between 13 and 30 percent of the children living in the contaminated areas had absorbed excessive amounts of lead (more than 40 micrograms per 100 milliliters of blood and more than 100 micrograms per gram of hair) as compared with less than 1 percent in a control group. A relationship between blood and hair was established which indicated that the absorption was fairly constant for most children examined. It seemed that the ingestion of contaminated dirt and dusts rather than "paint pica" was the major route of lead intake. Metabolic changes were found in most of 21 children selected from those with excessive lead absorption; 10 to 15 percent of this group showed subtle neurological dysfunctions and minor psychomotor abnormalities.*

Attempts to establish a dose-response relationship between environmental lead concentrations and subtle changes in human health have met with only limited success, largely because of the multiplicity of sources and the consequent difficulties of quantifying individual exposure. We report here the health effects of lead contamination around two secondary lead smelters.

Although smelters A and B are located in different parts of Toronto, each smokestack (in this report all distances are measured from the respective smokestack) is about 160 m south of a residential area and 100 to 200 m north of an elevated expressway (10 to 20 m high carrying 50,000 to 150,000 cars per day). Lead emissions from the two smelters were estimated to be 15,000 and 30,000 kg/year, respectively. Procedures for the collection, preparation, and analysis of materials have been described extensively elsewhere (*1*).

A mosaic of lead concentrations in the soil was found in each urban-industrial complex, but extremely high values were recorded in localized areas around each of the smelters (Fig. 1a). Regression analysis of the concentrations indicated that an exponential decrease with distance, from values of 40,000 and 16,000 µg per gram of soil close to smelter A and smelter B, respectively, to an urban background of 100 to 500 µg per gram of soil, accounted for 60 [illegible] percent of the variability in the [illegible]. The [illegible] monthly variation in the lead content of dustfall was [illegible] to the mean, but regression analysis also indicated an exponential decrease with distance from each smelter (Fig. 1b). In this case the distance parameter accounted for only 40 to [illegible] percent of the data variability within the limit of influence. Because the lead fallout was very localized and consisted primarily of large particles, it appears that the emissions originated mainly from low-level, dust-producing operations rather than from stack fumes.

Unlike the extremely high soil and dustfall values, the monthly geometric means of the lead concentrations in suspended particles close to the smelters (1 to 5.3 µg per cubic meter of air) were only double those for urban sites (0.8 to 2.4 $\mu g/m^3$). However, the range of daily concentrations was much greater, producing a marked log-normal distribution (Fig. 1c). The episodal peaks were correlated with winds from the smelters, but extensive monitoring was necessary to separate emissions from the smelters and auto emissions on the expressway when winds were from the south. At two sites the same distance from the expressway as samplers north of smelter A and smelter B (that is, 300 and 200 m, respectively), the lead concentrations averaged 1.0 and 1.6 $\mu g/m^3$, in agreement with calculations from values reported by Daines *et al.* (*3*). The lead aerosol was predominantly submicron in size, having a mass median diameter (MMD) of 0.8 ± 0.2 µm. This was also the distribution at sites close to the smelters except for episodal days when, for example, concentrations at one site 100 m north of smelter A averaged 6.6 ± 1.0 $\mu g/m^3$ and the MMD increased to 4.6 ± 1.3 µm. The average concentrations of antimony and arsenic, also associated with smelter fumes, increased from 20 ng/m^3 to 440 and 180 ng/m^3, respectively, on episodal days, and the size distribution reflected similar changes between nonepisodal and episodal days. Bromine concentrations, associated with automobile emissions, were about 0.3 $\mu g/m^3$, irrespective of wind direction with an MMD of 0.4 ± 0.2 µm. The average bromine/lead ratio at the control sites was 0.32 ± 0.04 as compared to 0.05 ± 0.02 close to smelter A on episodal days, which is a value similar to that found by Wesolowski *et al.* (*4*) for samples containing industrial lead emissions.

Between May and December 1973, the Toronto Board of Health collected capillary blood from 286 people close to smelter A, 1425 people close to smelter B, and 1231 people in a similar socioeconomic urban control group, and had these samples analyzed for lead content by anodic stripping voltammetry (5). The arithmetic mean for the control group was 19 μg per 100 ml of blood, and less than 1 percent of the children in this group (0 to 14 years) had lead contents in excess of 40 μg/100 ml. The means for children living withing 300 m of smelters A and B were 27 and 28 μg/100 ml, respectively. The log-normal distribution about these values was significantly higher than that of the control area on the basis of chi-square analysis ($p < .001$), mainly because 28 percent of the children sampled during the summer months close to smelter A and 13 percent sampled during the winter months close to smelter B had blood lead contents in excess of 40 μg/100 ml. Although there was no significant trend for the adults, the blood lead concentrations of the children increased with proximity to both smelters (Fig. 1d). However, only 30 and 8 percent of the variation in blood lead concentrations within 300 m of smelters A and B, respectively, could be attributed to the distance variable. Similar increases in the blood lead content of people living in contaminated areas have been found by other workers (6), although such increases were only marginal in old lead-mining areas (7).

In order to determine the major source of increased lead absorption, a detailed survey of 16 families (out of a possible 30) living in houses within 150 m of smelter A and 8 urban control families living in houses of similar age and upkeep was carried out between April and November 1973. The analy-

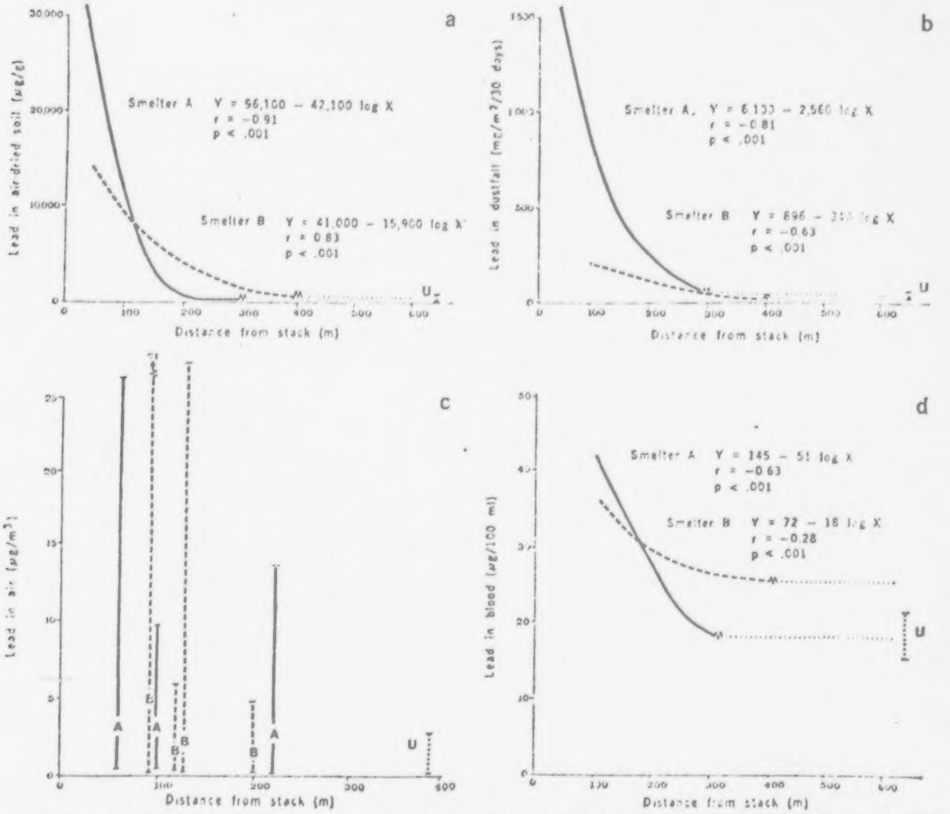

Fig. 1. Dispersal of lead contamination around two secondary smelters and accumulation by children. Solid lines and dashed lines in (a), (b), and (d) are fitted curves corresponding to the regression equations; dotted lines are an extrapolated fit. *U* indicates the corresponding values found in urban control areas; *A* and *B* in (c) are the geometric means ± range for 24-hour high-volume air samples close to smelter A and smelter B, respectively. In (a) the number of soil samples is 121 around smelter A and 146 around smelter B. In (b) the total number of monthly dustfall samples is 96 at 16 sites close to smelter A and 34 at 6 sites close to smelter B. In (c) the number of 24-hour air samples is 96, 57, and 94 at sites 60, 100, and 220 m from smelter A and 101, 64, 73, and 101 at sites 90, 120, 130, and 195 m from smelter B. In (d) the number of children is 84 close to smelter A (0 to 14 years) and 1041 close to smelter B (0 to 15 years).

sis of the lead content of capillary blood and entire lengths of head hair of the residents gave geometric means of 17 μg per 100 ml of blood and 13 μg per gram of hair in the control group as compared to 27 μg/100 ml and 41 μg/g for the smelter group (Table 1). The natural logarithm of the hair lead content was correlated with unaltered blood lead contents (correlation coefficient $r = 0.76$) for 68 of the 73 individuals sampled, which is in agreement with the results of Hammer *et al.* (*8*). Such a relationship might be expected if the rate of absorption is fairly constant. The observation that the lead content of hair segments did not vary appreciably with the distance of the segment from the scalp for children who fell into the correlated group strongly suggests that this is the case. The relationship does not hold true for children exposed to acute doses of lead (*9*), and it did not occur in three children of an occupationally exposed worker or two children with anecdotal evidence of soil pica.

The lead content of tap water was low in both study areas, the consumption of homegrown vegetables was not extensive, and, although the lead content of paint was extremely variable, there was no difference in the lead content of the house paint between smelter and control houses (Table 1). Suspended lead in air was higher close to the smelters than at a distance, but 70 percent of the increase was attributable to large nonrespirable particles; indoor values (during the winter months) were only 20 to 30 percent of those recorded outdoors. Ingestion of contaminated dirt and dusts may be the major route of intake by children (*10*), particularly in this case, as 20 to 60 percent of the lead in surface soil was extractable in 0.1*N* HCl compared with less than 10 percent extractable from paint samples.

To date, 21 of the children with excessive lead absorption have been admitted to the hospital for closer observation. Metabolic changes, as indicated by increased excretion of δ-aminolevulinic acid (ALA) and coproporphyrins (*11*), were observed in most of these cases. Increased density in bone metaphyses was observed in four children. The criteria used as indicating the need for chelation therapy (*12*) were urinary excretion of ALA in excess of 2 mg per square meter of body surface over a 24-hour period and urinary lead excretion in excess of 1 mg per gram of ethylenediaminetetraacetic acid (EDTA) over a 24-hour period, after mobilization with 25 mg of the calcium salt of EDTA per kilogram of body weight. The difficulty of determining a level of "significant toxicity" is shown by the fact that 14 cases exceeded the former criterion whereas five cases exceeded the latter. Two individuals exhibited neurological abnormalities of a nonspecific nature, and a reduction in peripheral nerve conduction velocity was recorded in three cases (*13*). Seven children were hyperactive, but it is still unclear whether lead is an etiological factor in, or the consequence of, hyperactivity (*14*). These findings are consistent with other studies (*15*), but the functional consequences of asymptomatic but elevated lead absorption [illegible]

We conclude that [illegible] hygiene [illegible] in areas where [illegible] content of house dust, outdoor dust, and soil is considerably elevated [illegible] in excessive lead absorption. The increases recorded do not appear sufficient to cause clear-cut symptoms of lead poisoning but may cause metabolic and subtle neurological changes.

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Table 1. Lead accumulation by people living close to a secondary smelter compared to an urban control group, and some potential sources of lead uptake. Abbreviations: A.M., arithmetic mean; G.M., geometric mean; D.W., dry weight; and F.W., fresh weight.

Matrix units	Smelter area			Urban control area		
	A.M.	G.M.	Range	A.M.	G.M.	Range
Blood (μg/100 ml)	31	27	12-61	19	17	8-40*†
Hair (μg/g)	52	41	14-166	17	13	3-39*†
Tap water (μg/liter)	6.8	5.9	2-14	1.40	1.3	1-2.4‖
Paint (% by weight)	1.23	0.72	0.13-4.64	1.30	0.89	0.11-2.62‡
Dustfall (mg/m² per 30 days)	553	158	35-2334	25	23	17-36*
Suspended particles (μg/m³)	3.84	3.01	1-10	0.93	0.82	0.30-2.54§
Outdoor dust (μg/g, D.W.)	5628	2416	578-39,500	1002	924	502-1561
House dust (μg/g, D.W.)	2055	1550	470-8400	845	713	351-2010
Garden soil (μg/g, D.W.)	2626	1715	355-8750	110	99	57-240*
Vegetables, washed						
Lettuce leaf (μg/g, F.W.)	8.6	2.9	0.7-40	0.9	1.0	0.8-1.1‡
Radish tuber (μg/g, F.W.)	4.6	1.7	0.6-28	0.3	0.3	0.2-0.4‡
Tomato fruit (μg/g, F.W.)	0.33	0.32	0.25-0.40	0.24	0.23	0.16-0.30‡

* Frequency distribution significantly different at the 0.1 percent level by Student's *t*-test after log-transformation. † Frequency distribution significantly different by chi-square analysis at the 0.1 percent level. ‡ Frequency distribution not significantly different. § Frequency distribution significantly different at 0.1 percent by paired comparison analysis of 24-hour air samples. ‖ Frequency distribution significantly different at the 5 percent level by Student's *t*-test after log-transformation.

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THE ASSOCIATION BETWEEN LEAD CONCENTRATIONS IN TEETH AND DOMESTIC WATER LEAD CONCENTRATIONS

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Summary

Tooth lead levels have been measured in 109 children living in Glasgow. A significant association has been demonstrated between molar tooth lead concentrations and domestic drinking water lead concentrations. Tooth lead concentrations were found to be greater the longer a child had lived in older housing with lead plumbing during foetal life and following birth. Tooth lead concentrations were also found to be age-related although no difference with respect to social class could be found.

Introduction

The use of dentine lead concentrations as a cumulative index of total lead exposure has excited some interest in recent years. Like bones, teeth accumulate lead. Over 90 percent of the body's lead burden is carried in bone, as was clearly demonstrated in the large post-mortem study carried out by Barry (1975) [1]. In the United States of America, Needleman et al. (1972) [2] found that the lead content of deciduous teeth of children living in urban areas was greater than those living in suburban areas. They attributed this difference to increased exposure to automobile exhaust containing lead. In circumstances where tooth and bone lead concentrations are elevated it is normally difficult, however, to ascribe these increased concentrations to any specific source. In recent studies it has been demonstrated that in the west of Scotland where water supplies are soft and where there are still considerable quantities of lead

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piping used for the distribution of domestic water supplies, there is a good association in adults between blood lead concentration and water lead concentration [3], and this has also been found to be the case in neonates. In view of this, the present study examined the association between tooth and lead concentrations and the concentration of lead in the domestic water supply of groups of children who had been exposed to lead for varying lengths of time since conception.

Materials and methods

Teeth were obtained from 109 children aged between 4 and 14 years presenting for routine dental care throughout Glasgow to Community Dental Health Officers, mainly within school clinics. A questionaire was completed at the time of tooth acquisition which included the child's age, the type of tooth (1st or 2nd molar) and parental occupation. At the same time details were taken of the home address occupied by the mother during pregnancy and the child during the first and second years of life, together with the current address. On the basis of the age and lead status of this housing and of the water lead values obtained from the domestic kitchen tap, the domestic lead exposure was divided into five basic groups. Group 1 consisted of children who had not been exposed to lead at any time from this source and had lived in the same house prior to and since birth. Groups 2, 3 and 4 were groups who had been exposed to lead but had subsequently moved to more modern housing with low exposure. Group 2 had been exposed to high water lead during the mother's pregnancy, Group 3 had been exposed to high water lead during the mother's pregnancy and the first year of life, and Group 4 had been exposed to high water lead during the mother's pregnancy, the first and second year of life. Group 5 had lived in the same older housing since birth and during pregnancy and their water leads were all in excess of 0.4 µmol/l.

Tooth lead concentrations were determined on molar teeth. All caries and amalgam fillings were carefully removed with a dental bur and the whole tooth washed and scraped in a 1% v/v solution of Decon 90 to remove as much tissue as possible. The tooth was then thoroughly rinsed in distilled water and dried. It was then weighed in a beaker and digested in 5 ml concentrated boiling nitric acid (Aristar BDH) until the solid remains contained no organic material. The digestion was completed in a muffle furnace at 380°C for six hours to remove all nitric acid. The tooth ash was weighed and dissolved in 10 ml 2 M hydrochloric acid (Aristar BDH) and the lead concentration measured on a polarograph (Shandon Southern A1660) against standards.

Water lead concentrations were measured by flameless atomic absorption spectrophotometry (Perkin Elmer 306 and HGA 72). It proved possible only to measure water leads in present housing in groups 2, 3 and 4, because of the small numbers of initial houses still extant following extensive demolition in Glasgow.

All samples were collected into lead-free containers as described previously [34]. Statistics were carried out by Mann Whitney U analysis and Spearman and Kendall ranking analysis because of the non-normal distribution of results. All figures were expressed in S.I. units, µmol lead/kg tooth ash and µmol lead/l domestic water.

Results

The numbers of children in each group, the mean age and the mean molar tooth lead and the mean domestic water lead are shown in Table I. Significance by Mann Whitney U analysis demonstrates significant elevations of tooth leads for the groups 5 (old housing), 4 and 3 (mixed housing) as compared with group 1 (new housing). There was no significant difference with group 2 (mixed-low exposure). Similarly, the means of tooth lead in groups 1, 2 and 3, but not in 4, were significantly less than in group 5. Water lead concentration of groups 2, 3 and 4 (mixed) were not significantly different to group 1, although group 5 water leads were significantly higher than those in group 1. No difference was found in the mean age or in the social class distribution for each of the groups (Table II) or in the tooth lead concentrations between the 1st and 2nd molars in each of the groups (Table III).

On consideration of the geometric means for each of the groups (Fig. 1)

TABLE I

TOOTH ANALYSIS OF 109 CHILDREN, 57 BOYS AND 52 GIRLS

Group 1, new housing throughout; 2, old housing in pregnancy, moving to new housing; 3, old housing in pregnancy, and first year of life, moving to new housing; 4, old housing in pregnancy, and first and second years of life, moving to housing; 5, old housing throughout.

	Group No.				
	1	2	3	4	5
No. of children	39	11	8	32	19
Mean age (±S.D.)	7.9 (±2.5)	8.3 (±2.9)	9.1 (±2.9)	9.2 (±2.5)	8.3 (±2.7)
Mean ± S.D. molar tooth lead (μmol/kg ash)	50 ± 33 †	65 ± 30 ***	76 ± 27 *,†	116 ± 83 **	121 ± 51 **
Mean ± S.D. domestic water lead (present home) (μmol/l)	0.16 ± 0.12	0.18 ± 0.15	0.27 ± 0.19	0.20 ± 0.12	1.65 ± 2.16 **

* $P < 0.02$ with respect to group 1.
** $P < 0.001$ with respect to group 1.
*** $P < 0.05$ with respect to group 5.
† $P < 0.001$ with respect to group 5.

TABLE II

SOCIAL CLASS DISTRIBUTION FOR THE CHILDREN IN THE VARIOUS GROUPS, PERCENTAGE OF EACH GROUP IN BRACKETS

Group	Social class				
	1	2	3	4	5
1	–	2 (5.1)	8 (20.5)	16 (41.0)	13 (33.3)
2	–	1 (9.0)	2 (18.2)	5 (45.5)	3 (27.3)
3	–	1 (12.5)	2 (25.0)	2 (25.0)	3 (37.5)
4	–	2 (6.3)	7 (21.9)	12 (37.5)	11 (34.4)
5	1 (5.3)	–	4 (21.1)	5 (26.3)	9 (47.3)

TABLE III

DIFFERENCE BETWEEN 1st AND 2nd MOLAR TEETH AND NUMBERS OF PERMANENT TEETH

Group No.	No. of permanent teeth	Molar type	No. of teeth	Mean (±S.D.) tooth lead (μmol/kg ash)
1	4	1	16	47 ± 22
		2	23	48 ± 32
2	0	1	6	58 ± 25
		2	5	76 ± 37
3	0	1	3	90 ± 27
		2	5	68 ± 27
4	3	1	17	100 ± 64
		2	15	128 ± 98
5	2	1	9	88 ± 30
		2	10	139 ± 63

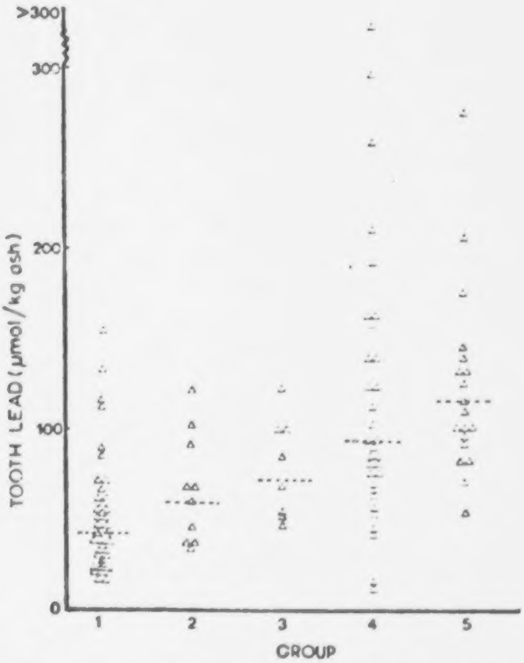

Fig. 1. Tooth lead concentrations in five groups of children in Glasgow. The dotted line shows the geometric mean for each of the groups. Group 1 are those children who had lived in new housing since birth and prior to birth; Group 2 were children who had been exposed to high water lead during the mother's pregnancy; Group 3 had been exposed to high water lead during the mother's pregnancy and the first year of life; Group 4 were children who had been exposed to high water lead during the mother's pregnancy, the first and the second year of life. Group 5 were those children who had lived in the same older housing since birth and during pregnancy.

(because of the 'tail' in the distribution of tooth leads) a gradation of values is shown to rise consistently as the degree of exposure to lead rises. In group 1 (new housing) the exposure to water lead was sufficiently consistent to allow an analysis of the association between age and tooth lead. A significant correlation was found between these two parameters by Spearman and Kendall ranking analysis ($P < 0.02$). The significance of this association was, however, less than the linear regression of tooth lead on water lead for groups 1 and 5, that is, those who had lived in the same housing throughout, for which $r = 0.73$ ($P < 0.001$), of equation

tooth lead = 50 water lead + 40.6

Since the age distributions of all 5 groups were similar, as were the mean ages, age was not considered to have influenced the results. The results of tooth lead are here calculated as per kg tooth ash, because of the greater consistency of results thus obtained. For fourteen teeth, weights of whole teeth and of their ash were obtained. The mean ratio of whole tooth weight to tooth ash weight was found to be 1.235 ± 0.163 (S.D.). A total of 9 permanent teeth were included in the study. These were evenly distributed through the groups in relation to their size (Table III). No significant difference could be found on Mann-Whitney U analysis between the 1st and 2nd molar teeth for each of the groups, nor was there an overall difference between them, although there was an apparent rise in the 2nd molar teeth in group 5 (Table III).

Discussion

The present study has demonstrated an association between lead concentrations of domestic drinking water supplies and the tooth lead concentrations of children brought up in houses supplied with this water. The evidence for actual exposure to lead from these homes is both as measured and inferred water lead concentrations. For about half of the children in this study, that is those who had changed habitation following birth, it proved impossible to identify a sufficient number of homes that had not been demolished. In addition, it was impossible to be certain that plumbing alterations had or had not been carried out. In consequence, only the water supply of existing housing was examined. It is clear, however, both from the figures given for the older houses in this study and from findings of previous studies in Glasgow [4,5,6], that such older houses do have considerably elevated water lead concentrations as a consequence of the quantity of lead water piping and lead-lined storage tanks within the house. Indeed the difference between the arithmetic mean water lead concentration for the new and old houses in this study (Groups 1 and 5) is about tenfold and is identical to that found previously in Glasgow [4]. This association between older housing, water lead and increased tooth lead concentrations is consistent with the studies of Stewart [7], who identified areas of Belfast with older housing, lead plumbing and increased tooth lead in children.

Previous studies have shown that dentition is a good indicator, like bone, of past exposure to lead [2,7–14]. Indeed, such lead accumulation is dose-related and permanent within the tooth [15]. The uptake of lead into teeth and bone takes place during formation from the systemic circulation. There is the possibility that some uptake might occur after eruption directly from the mouth. However, the work of Steadman et al. [16] does not support that view. In these various studies the type of exposure to lead has varied considerably but has been ascribed to pica (such as chewing printed paper) [17], living in deteriorating housing with lead paint [10,11], industrial emissions [18] and urban air lead concentrations [2].

As shown in the present study, tooth lead concentrations are age-related. This has been demonstrated before [2,7,19,20,21], and indeed would be expected to be so if such exposure is dose-related. Holtzman et al. [33] have suggested that such age-related rises plateau towards the end of the third decade. Others have shown greater concentrations of lead in deciduous teeth [35]; the numbers analysed in the present study were too few to confirm this. Some authors [19,22,23], have identified the highest concentrations within the tooth as being in the circumpulpal dentine zone. Others have shown that concentrations vary across the enamel [24]. It has also been shown that tooth lead concentrations vary [11,15] dependent upon tooth type, with the greatest concentration in the incisors and least in the molars [25], although other workers did not find this to be so [26] and we were unable to demonstrate differences between first and second molar teeth. Strehlow and Kneip [20], have shown that tooth lead concentrations were six times greater than skeletal bone lead concentrations. We did not attempt to dissect specific areas of teeth in this study, but rather preferred to use whole molar teeth. The range of values arrived at in this study (16–400 μmol/kg ash) are similar to those previously published, when corrected for tooth weight (20–494 μmol/kg tooth). Thus, in their recent survey in Birmingham Mackie et al. [25] gave a range of 4–522 μmol/kg tooth and listed a series of similar values from other publications.

Geographical circumstances can clearly vary tooth lead concentrations [2,23, 27], and urban situations are usually associated with higher concentrations in teeth than in suburban or rural situations [7,18,25,28,29]. However, as shown by Lockeretz [15] and Mackie et al. [25], tooth lead levels are associated in industrial urban situations with generalised lead exposure and cannot usually be attributed to one specific source, although Stack et al. [18] were able to associate such exposure with a lead smelter. The children examined in this study derived from urban environments within the city of Glasgow.

Tooth lead concentrations have been measured in the past by various methods including mass spectrometry [30], activation analysis [31], electron probe analysis [32], atomic absorption spectrophotometry [7,15], and anodal stripping voltammetry [22]. The present method of cathode ray polarography has the advantage of both sensitivity and specificity, especially in the presence of high concentrations of calcium and other ions that might interfere with analysis. The present method does have the disadvantage of slow through-put because of the necessity for initial digestion of samples.

The results shown here do not exclude the contribution of other sources of lead exposure such as pica or lead in the air to tooth lead concentrations and, therefore, to the total body lead burden. For the children studied, it would appear that increased domestic water lead concentrations are contributing significantly to the total body lead burden of these children, as evidenced by the association between water lead and tooth lead concentrations in a fashion similar to that demonstrated between blood lead and water lead [3].

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federal register

THURSDAY, OCTOBER 5, 1978
PART IV

THE NATIONAL ARCHIVES OF THE UNITED STATES • 1934 •

ENVIRONMENTAL PROTECTION AGENCY

NATIONAL AMBIENT AIR QUALITY STANDARD FOR LEAD

Final Rules and Proposed Rulemaking

[6560-01]

Title 40—Protection of Environment

CHAPTER I—ENVIRONMENTAL PROTECTION AGENCY

SUBCHAPTER C—AIR PROGRAMS

[FRL 937-5]

PART 50—NATIONAL PRIMARY AND SECONDARY AMBIENT AIR QUALITY STANDARDS

National Primary and Secondary Ambient Air Quality Standards for Lead

AGENCY: Environmental Protection Agency.

ACTION: Final rulemaking.

SUMMARY: EPA is setting a national ambient air quality standard for lead at a level of 1.5 micrograms lead per cubic meter of air (μg Pb/m^3), averaged over a calendar quarter. This final rulemaking follows a 1976 court order to list lead as a criteria pollutant for the development of an ambient standard, and the Agency's issuance of a proposed standard on December 14, 1977. In response to comments received on the proposed standard, EPA has changed the averaging period for the standard from a calendar month to a calendar quarter, and has clarified the health basis used in selecting the standard level.

In establishing the level of the final standard, EPA has determined that young children (age 1-5 years) should be regarded as a group within the general population that is particularly sensitive to lead exposure. The final standard for lead in air is based on preventing most children in the United States from exceeding a blood lead level of 30 micrograms lead per deciliter of blood (μg Pb/dl). Blood lead levels above 30 μg Pb/dl are associated with the impairment of heme synthesis in cells indicated by elevated erythrocyte protoporphyrin (EP), which EPA regards as adverse to the health of chronically exposed children. There are a number of other adverse health effects associated with blood lead levels above 30 μg Pb/dl, in children as well as in the general population, including the possibility that nervous system damage may occur in children even without overt symptoms of lead poisoning.

DATES: Effective: October 5, 1978. After promulgation, States will have nine months (until July 5, 1979), to prepare and submit to EPA plans for attainment of the standard by no later than October of 1982. EPA's final regulations for the development of State implementation plans appear elsewhere in this FEDERAL REGISTER.

FOR FURTHER INFORMATION CONTACT:

Mr. Joseph Padgett, Director, Strategies and Air Standards Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, N.C. 27711, telephone 919-541-5204.

AVAILABILITY OF RELATED INFORMATION

A docket (No. OAQPS-77-1) containing the information used by EPA in the development of the proposed standard is available for public inspection and copying between 8 a.m. and 4:30 p.m., Monday through Friday, at EPA's Central Docket Section, Room 2903B, Waterside Mall, 401 M Street SW., Washington, D.C. 20460.

The Federal reference method for collecting and measuring lead and its compounds in the ambient air is published in appendix G to this promulgation. This FEDERAL REGISTER also contains proposed regulations under 40 CFR Parts 51 and 53 for equivalent lead air monitoring methods, final rules for the development of State implementation plans promulgated under 40 CFR Part 51, and an advance notice of proposed rulemaking under 40 CFR Part 51 for ambient monitoring in the vicinity of certain industrial plants with lead emissions. Additional information for the development of the State implementation plans is contained in the document Supplementary Guidelines for Lead Implementation Plans. The environmental and economic impacts of implementing this standard are described in an environmental impact statement and an economic impact assessment. These documents are available for public inspection and copying at the Central Docket Section (address above). Copies may be obtained upon request from Mr. Joseph Padgett at the above address.

The documents Air Quality Criteria for Lead and Control Techniques for Lead Air Emissions were issued at the time of proposal. The Control Techniques Document is available upon request from Mr. Joseph Padgett at the above address. The Air Quality Criteria Document can be obtained from: Mr. Michael Berry, Environmental Criteria and Assessment Office, MD-52, Office of Research and Development, U.S. Environmental Protection Agency, Research Triangle Park, N.C. 27711, telephone 919-541-2266.

SUPPLEMENTARY INFORMATION:

BACKGROUND

Lead is emitted to the atmosphere by vehicles burning leaded fuel and by certain stationary sources. Lead enters the human body through ingestion and inhalation with consequent absorption into the bloodstream and distribution to all body tissues. Clinical, epidemiological, and toxicological studies have demonstrated that exposure to lead adversely affects human health.

EPA's initial approach to controlling lead in the air was to limit the lead emissions from automobiles, the principal source of lead air emissions. Regulations for the phasedown of lead in the total gasoline pool were promulgated in 1973, and, following litigation, modified and put into effect in 1976. The Agency has also established regulations requiring the availability of no-lead gasoline for catalyst-equipped cars. EPA also intended to control emissions from certain categories of industrial point sources under section 111 of the Clean Air Act.

In 1975, the Natural Resources Defense Council (NRDC) and others brought suit against EPA to list lead under section 108 of the Clean Air Act as a pollutant for which air quality criteria would be developed and a national ambient air quality standard established under section 109 of the Act. The Court ruled in favor of NRDC. (*NRDC, Inc. et al. v. Train*, 411 F. Supp. 864 (S.D.N.Y., 1976) aff'd 545 F. 2d 320 (2d Cir. 1976).) EPA listed lead on March 31, 1976, and proceeded to develop air quality criteria and the proposed standard.

On December 14, 1977, EPA proposed a standard of 1.5 μg Pb/m^3, calendar month average, proposed the Federal reference method for monitoring air lead levels, issued the documents Air Quality Criteria for Lead and Control Techniques for Lead Air Emissions and proposed regulations for State implementation plans. EPA invited public comments during the period from December 14, 1977, to March 17, 1978, on the standard, reference method, and the SIP regulations. Additional comments on these matters were provided to EPA at a public hearing held on February 15-16, 1978.

LEGISLATIVE REQUIREMENTS FOR NATIONAL AMBIENT AIR QUALITY STANDARDS

Sections 108 and 109 of the Clean Air Act govern the development of national ambient air quality standards. Section 108 instructs EPA to document the scientific basis for the standard:

> Section 108(a)(2). The Administrator shall issue air quality criteria for an air pollutant within 12 months after he has included such pollutant in a list under paragraph (1). Air quality criteria for an air pollutant shall accurately reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the presence of such pollutant in the

> ambient air, in varying quantities. The criteria for an air pollutant, to the extent practicable, shall include information on—
>
> (A) Those variable factors (including atmospheric conditions) which of themselves or in combination with other factors may alter the effects on public health or welfare of such air pollutant;
>
> (B) The types of air pollutants which, when present in the atmosphere, may interact with such pollutant to produce an adverse effect on public health or welfare; and
>
> (C) Any known or anticipated adverse effects on welfare.

Section 109 addresses the actual setting of the standard:

> Section 109(b)(1). National primary ambient air quality standards, prescribed under subsection (a) shall be ambient air quality standards the attainment and maintenance of which in the judgment of the Administrator, based on such criteria and allowing an adequate margin of safety, are requisite to protect the public health. Such primary standards may be revised in the same manner as promulgated.
>
> (2) Any national secondary ambient air quality standard prescribed, under subsection (a) shall specify a level of air quality the attainment and maintenance of which in the judgment of the Administrator, based on such criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of such air pollutant in the ambient air. Such secondary standards may be revised in the same manner as promulgated.

In order to conform to the requirements of section 109, EPA has based the level of the lead air quality standard on information presented in the criteria document pertaining to the health and welfare implications of lead air pollution. This is in contrast to other sections of the Act under which EPA considers economic costs and technical availability of air pollution control systems in determining emissions limitations. It is clear from section 109 that the Agency should not attempt to place the standard at a level estimated to be at the threshold for adverse health effects, but should set the standard at a lower level in order to provide a margin of safety. EPA believes that the extent of the margin of safety represents a judgment in which the Agency considers the severity of reported health effects, the probability that such effects may occur, and uncertainties as to the full biological significance of exposure to lead.

Comments resulting from external review of the air quality criteria and the proposed standard highlight disagreements on a number of areas critical to EPA's rationale for the standard. However, the scientific data base provided in the document Air Quality Criteria for Lead is as extensive as that for any other regulated air pollutant. Also, at every stage of development of the air quality criteria and the standard, EPA has facilitated and received broad external participation. EPA regards as inevitable the presence of scientific disagreement and uncertainty about key factors relevant to environmental standards. Provisions of the Act requiring timely promulgation of the standard, and requirements for periodic future review of air quality criteria and standards indicate congressional intent that the Agency proceed even where scientific knowledge is not complete or full scientific consensus is absent.

SUMMARY OF GENERAL FINDINGS FROM AIR QUALITY CRITERIA FOR LEAD

Following the listing of lead as a criteria pollutant, EPA developed the document, Air Quality Criteria for Lead. In the preparation of this document, EPA provided opportunities for external review and comment on three successive drafts. The document was reviewed at three meetings of the Subcommittee on Scientific Criteria for Environmental Lead of EPA's Science Advisory Board. Each of these meetings was open to the public and a number of individuals presented both critical review and new information for EPA's consideration. The final criteria document was issued on December 14, 1977.

From the scientific information in the criteria document, EPA draws conclusions in several key areas with particular relevance for the ambient air quality standard for lead.

1. There are multiple sources of lead exposure. In addition to air lead, these sources include: Lead in paint and ink, lead in drinking water, lead in pesticides, and lead in fresh and processed food.

2. Exposure to air lead can occur directly by inhalation, or indirectly by ingestion of lead contaminated food, water, or nonfood materials including dust and soil.

3. There is significant individual variability in response to lead exposure. Even within a particular population, individual response to lead exposure may vary widely from the average response for the same group. Certain subgroups within the general population are more susceptible to the effects of lead or have greater exposure potential. Of these, young children represent a population of foremost concern.

4. Three systems within the human body appear to be most sensitive to the effects of lead—the blood-forming or hematopoietic system, the nervous system, and the renal system. In addition, lead has been shown to affect the normal functions of the reproductive, endocrine, hepatic, cardiovascular, immunologic, and gastrointestinal systems.

5. The blood lead level thresholds for various biologic effects range from the risk of permanent, severe, neurological damage or death as blood leads approach and exceed 80 to 100 μg Pb/dl in children down to the inhibition of an enzymes system as low as 10 μg Pb/dl.

6. Lead is a stable compound, ubiquitously distributed, which persists and accumulates both in the environment and in the human body. In developing the proposed standard, EPA used these findings to arrive at a standard level of 1.5 μg Pb/m^3, calendar month average. This level was derived from the Agency's judgment that the maximum safe blood lead level (geometric mean) for a population of young children was 15 μg Pb/dl and, of this amount, 12 μg Pb/dl should be attributed to nonair sources. The difference of 3.0 μg Pb/dl was estimated to be the allowable safe contribution to mean population blood lead from lead in the air. With epidemiological data indicating a general relationship of 1:2 between air lead (μg Pb/m^3) and blood lead (μg Pb/dl), EPA determined that the level for the proposed standard should be 1.5 μg/m^3.

SUMMARY OF ANTICIPATED IMPACTS

While the level of the standard is based on health considerations, EPA has conducted economic and environmental studies to assess the potential impacts of the standard selected. EPA estimates that the existing regulations for the phase-down of lead in gasoline, combined with the increasing use of no-lead gasoline for catalyst-equipped cars, will result in attainment of the standard in urban areas where automobile exhaust is the dominant source of air lead. No additional pollution controls are anticipated for these areas.

EPA's economic analysis does indicate that there may be significant problems in attainment of the standard in the vicinity of nonferrous smelters and other large industrial sources of lead emissions. This assessment is based, however, on studies using general emission factors and plant configurations, combined with dispersion modeling. In the development of State plans to implement the standard, EPA is encouraging affected industries and State agencies to gather plant-specific technical data, ambient air quality data, and assessments of alternative engineering controls. With this information, the Agency will be able to more accurately evaluate the impact of the standard and better consider approval of alternative approaches to emission control in the State plans.

Also, EPA is encouraging affected firms and State agencies to evaluate in the early design phase, strategies which take into consideration the workplace standard for airborne lead which will be promulgated by the Occupational Health and Safety Admintration (OSHA). EPA believes that his approach will facilitate applicaon of control technologies which neet the requirements of both agenes. In working with OSHA to estinate the combined impact of the SHA and EPA standards, in coordiating compliance strategies, and in eviewing State plans implementing he ambient standard, EPA intends to void an approach which would foster ncertainty in the investment decions of affected firms.

The Agency will make every effort o insure that all opportunities to void plant closures are examined, hile at the same time assuring proction from clear risks to the public ealth.

SUMMARY OF COMMENTS RECEIVED

During the comment period from ecember 14, 1977, to March 17, 1978, nd at the public meeting on February 5-16, 1978, EPA received 95 written nd oral comments addressing the proosed standard or the requirements or State implementation plans. All omments opposing the standard as xcessively stringent (25) came from epresentatives of affected industries, nd 20 of these counter-proposed 5.0 g Pb/m^3, calendar quarter average, as he appropriate level for the standard.

omments Received Opposing the Proposed standard of 1.5 $\mu g/m^3$ Calendar Month Average as Excessively Stringent

Company	Opposed[1]	Endorsed[2]
max Lead & Zinc, Inc	X	X
merican Mining Congress	X	X
merican Petroleum Institute	X	
SARCO	X	X
ssociated Octel Co., Ltd	X	X
attery Council International	X	X
ethlehem Steel Corp	X	X
unker Hill Co	X	X
& D Batteries Division	X	X
I. du Pont de Nemours & Co., Inc	X	X
SA Laboratories, Inc	X	X
thyl Corp	X	X
eneral Battery Corp	X	X
eneral Motors Corp		
etty Refining & Marketing Co	X	
ECLA Mining Co	X	
ouston Chemical	X	X
unt Oil Co	X	
err McKee Corp		
ead Industries Association	X	X
alco Chemical	X	X
L Industries, Inc	X	X
restolite Battery Division	X	X
econdary Lead Smelters Association	X	X
hell Oil Co	X	
Joe Minerals Corp	X	X
exaco, Inc	X	X
nited Machinery Group		
ulcan Materials Co		

[1] 1.5 $\mu g/m^3$, calendar month.

[2] 5.0 $\mu g/m^3$, calendar quarter (or other averaging riod).

SUMMARY: Forty-five comments received from 29 rporations or their representatives. 25 of the 29 rms opposed the proposed standard of 1.5 $\mu g/m^3$, alendar month average. 20 endorsed an alternative standard of 5.0 $\mu g/m^3$ calendar quarter average (or other averaging period).

Four comments opposed the proposed standard on the grounds that it was not sufficiently protective of health.

COMMENTS RECEIVED OPPOSING PROPOSED LEAD AIR QUALITY STANDARD OF 1.5 $\mu g/m^3$, CALENDAR MONTH AVERAGE, IN FAVOR OF A MORE STRINGENT STANDARD

- Natural Resources Defense Council
- Dr. Sergio Piomelli, Director, Pediatric Hematology, New York University Medical Center Public Interest Campaign
- University of Connecticut School of Medicine

Comments supporting the level of the proposed standard (17) came from the medical community, Federal agencies, State and local public health agencies, and public interest groups.

COMMENTS RECEIVED ENDORSING PROPOSED LEAD AIR QUALITY STANDARD OF 1.5 $\mu g/m^3$, CALENDAR MONTH AVERAGE

STATE AND LOCAL AGENCIES

- California Department of Health
- Massachusetts Department of Public Health
- New York State Department of Environmental Conservation
- New York City Department of Environmental Protection
- Tennessee Department of Public Health
- Wisconsin Department of Natural Resources

FEDERAL AGENCIES

- Center for Disease Control, Public Health Service
- Department of Transportation
- Food and Drug Administration
- Occupational Safety and Health Administration

PUBLIC INTEREST GROUPS AND THE MEDICAL COMMUNITY

- Committee on Environmental Hazards, American Academy of Pediatrics
- D.C. Committee for Lead Elimination in the District
- League of Women Voters of the United States
- National Urban League
- Herbert Needleman, Boston Children's Hospital Medical Center
- University of North Carolina School of Public Health

In addition, EPA has received numerous comments and correspondence on the proposed standard after the official end of the comment period. Though EPA does not have a legal obligation to review these documents, it has, in the interest of fostering full public participation in the rulemaking process, reviewed these comments and correspondence as time permitted. As with all other documents considered or examined by EPA as part of its decision process, these documents have been placed in the public docket and have become part of the administrative record of this decision.

The comments received by EPA did not challenge three aspects of the proposed standard:

1. The basic structure of the rationale used by the Agency in deriving the level of the proposed standard.
2. The selection of young children as a population particularly at risk to lead exposure.
3. The attribution of 12 μg Pb/dl out of the target mean population blood lead level of 15 μg Pb/dl to nonair sources of lead for the purposes of setting the air standard.

Significant comments were received, however, on the following key areas relating to the standard:

1. The elevation of erythrocyte protoporphyrin (EP) as the first adverse health effect with increasing lead exposure rather than the decline of hemoglobin levels.
2. The blood lead threshold level for elevated EP.
3. The incidence of health effects in populations residing in the vicinity of industrial sources of lead particulate emissions.
4. The relationship describing the response of lead in the blood to lead in the air.
5. The statistical form and averaging period for the standard.
6. The appropriate margin of safety.
7. The limitation of the standard to the respirable fraction of total air lead particles.
8. The economic impact of the standard.
9. The State implementation plan regulations.
10. The Federal reference method for monitoring lead air quality.
11. The administrative procedures employed by EPA in the development of the standard and the provision for public participation.

A review of the comments received and their disposition has been placed in the rulemaking docket (OAQPS-77-1) for public inspection. The following paragraphs summarize the significant comments and present the Agency's findings.

THE HEALTH SIGNIFICANCE OF ERYTHROCYTE PROTOPORPHYRIN ELEVATION

Ten commenters disagreed with EPA's conclusion that the impairment of heme synthesis indicated by elevated erythrocyte protoporphyrin (EP) constituted an adverse health effect. Reasons for this disagreement included:

1. An elevated level of EP is not itself toxic to the cells in blood or other tissues.
2. EP elevation, while indicating a change in heme synthesis, does not indicate an insufficient production of heme, or hemoglobin.
3. EP elevation and the alteration of heme synthesis does not imply impair-

ment of other mitochondrial functions.

4. EP elevation is not associated with impairment of other heme proteins, particularly cytochrome P-450.

5. Elevated EP may be caused by conditions other than exposure to lead, particularly iron deficiency.

Five commenters agreed with EPA's conclusions about the health significance of elevated EP citing the following arguments:

1. The interference of lead in a fundamental cellular metabolic function to the extent that there is accumulation of a substrate is physiological impairment, even without the presence of clinical evidence of disease.

2. It is prudent medical practice to intervene where subclinical indicators of physiological impairment are present.

3. The impairment of heme synthesis resulting from genetic or dietary factors places a child at enhanced risk to lead exposure.

4. There is evidence to suggest that impaired heme synthesis may affect the function of neural or hepatic tissue even at levels where heme production is sufficient for hematopoiesis.

AGENCY RESPONSE

EPA agrees with the comments received that the initial elevation of EP as a result of exposure to lead, while indicating an impairment of heme synthesis, may not be a disease state or be seen as a clinically detectable decline in performance. However, the criteria document points out (p. 1-13) that this impairment does increase progressively with lead dose.

> The hematological effects described above are the earliest physiological impairments encountered as a function of increasing lead exposures as indexed by blood lead elevations; as such, those effects may be considered to represent critical effects of lead exposure. Although it may be argued that certain of the initial hematological effects (such as ALAD inhibition) constitute relatively mild, nondebilitating symptoms at low blood lead levels, they nevertheless signal the onset of steadily intensifying adverse effects as blood lead elevations increase. Eventually, the hematological effects reach such magnitude that they are of clear-cut medical significance as indicators of undue lead exposure.

The fact that other conditions, such as iron deficiency may also impair heme synthesis, does not obviate concern that lead is interfering with an essential biological function. There is the possibility that a nutritional deficiency is an additional stress to the heme synthetic system which may increase the sensitivity of a child to the adverse effects of lead exposure.

EPA notes that there is general agreement that heme and heme-containing proteins play important roles in the oxygen fixation pathways in all cells. While the effects of low-level lead exposure on the heme synthetic pathway in erythroid tissue have been extensively studied in part because of the ease with which this tissue may be obtained, other cellular metabolic systems utilizing heme are less well understood. EPA does not have sufficient information to conclude that impairment of heme synthesis in other tissues is not of concern until blood lead levels are reached greater than those associated with hematological effects. The air quality criteria document does point out that this effect has been established in other tissues and that other dose-response factors may apply.

> The effect of lead on the formation of heme is not limited to the hematopoietic system. Experimental animal studies have shown a lead effect on the heme-requiring protein, cytochrome P-450, an integral part of the hepatic mixed-function oxidase (chapter 11), the systemic function of which is detoxification of exogenous substances. Heme synthesis inhibition also takes place in neural tissue. (P. 13-5.)

In summary, the criteria document states:

> Elevation in protoporphyrin is considered not only to be a biological indicator of impaired mitochondrial function of erythroid tissue but also an indicator of accumulation of substrate for the enzyme ferrochelatase. It therefore has the same pathophysiological meaning as increased urinary δ-ALA (vide supra). For these reasons, accumulation of protoporphyrin has been taken to indicate physiological impairment in humans, and this clinical consensus is expressed in the 1975 Statement of the Center for Disease Control (CDC), USPHS. The criterion used by CDC to indicate an effect of lead on heme function is an FEP level of 60 µg/dl in the presence of a blood lead level above 30 µg/dl whole blood.
>
> More recent information relating to threshold of lead effects indicates that FEP levels begin to increase at a blood lead value of 15 to 20 µg Pb/dl blood in children and women and, at a somewhat higher value, 20 to 25 µg Pb/dl blood, in adult men. (P. 13-8.)

EPA concludes that the state of elevated EP must be regarded as potentially adverse to the health of young children. While the onset or a mild experience of this condition may be tolerated by an individual, as with other subclinical manifestations of impaired function, it is a prudent public health practice to exercise corrective action prior to the appearance of clinical symptoms. The criteria document reports that symptoms of anemia in children may occur at blood lead levels of 40 µg/dl. EPA has adopted 30 µg Pb/dl as a maximum safe blood lead level for individual children.

THE BLOOD LEAD THRESHOLD FOR ELEVATED ERYTHROCYTE PROTOPORPHYRIN

Comments provided by ten organizations challenged EPA's conclusion that the threshold for the elevation of EP occurs in children at a blood lead level of 15 µg/dl. Evidence offered for a higher threshold included:

1. The threshold accepted by EPA is based on a study in which an inappropriate statistical technique, probit analysis, was employed.

2. Application of a more appropriate technique, segmented line analysis, results in a higher threshold.

3. The study in question excluded data on children with blood lead levels in excess of 30 µg/dl.

4. Other investigators have reported higher thresholds.

Comments in support of the 15 µg/dl threshold maintained:

1. It is proper to exclude values considered abnormal if the intent of the analysis is to determine an unbiased effect threshold.

2. Other studies have reported thresholds with error bands which include 15 µg/dl.

3. Probit analysis is an appropriate technique and differs only slightly from the results obtained from segmented line analysis.

AGENCY RESPONSE

EPA agrees that the segmented line technique provides a more accurate estimate of the correlation threshold of EP elevation with increasing blood lead, about 16.7 µg Pb/dl, and for this reason considered changing its judgments as to the maximum safe blood lead level for a population of children. However, as the target geometric mean for a population is increased, a greater percentage of children in the population will exceed the maximum safe individual level of 30 µg Pb/dl. EPA estimates that at a population geometric mean of 15 µg Pb/dl, 99.5 percent of children will be below 30 µg Pb/dl. At 16.7 µg Pb this percentage falls to 98.7. EPA regards the number of children predicted to be below 30 µg Pb/dl as the critical health consideration. For this reason, EPA has maintained its estimate of a geometric mean of 15 µg Pb/dl as the target for population blood lead.

THE INCIDENCE OF HEALTH EFFECTS IN POPULATIONS RESIDING IN THE VICINITY OF INDUSTRIAL SOURCES OF LEAD PARTICULATE EMISSIONS

Several comments cited situations in which proximity to significant point sources of airborne lead emissions appear to have little or no health impact on resident populations. This was taken to imply that the air standard was not necessary to protect public health.

AGENCY RESPONSE

EPA acknowledges the variability of the impact of exposure to air lead on the potential for adverse health consequences. It is clear that direct exposure to air lead is only one of the

ts through which human expo
o: curs. It is for this reason that
Agency has accepted the concept
only a portion of the : .fe popula-
mean blood lead level should be
ibutable to air lead exposure. The
ence or absence of health effects
n exposed population is influenced
: variety of factors including: Me-
ology, terrain characteristics, geo-
'al and anthropological history,
onal and domestic hygiene, the oc-
ıtions of the population members,
the food and nonfood materials
ı which they come into contact.
ing into account such variability, it
ains the Agency's belief that air-
ie lead directly and indirectly con-
ıtes to the risk of adverse health
equences and that sufficient clini-
and epidemiological evidence is
lable to form a judgment as to the
nt of this contribution. This evi-
e includes epidemiological studies
ving higher blood lead levels in
n areas where air lead levels were
ited in comparison to rural areas.
e have also been a number of
ies linking elevated blood lead
's to industrial sources of lead
sions. With regard to the 1972
y at El Paso, Tex., by the Center
Disease Control, the criteria docu-
t reports:

> was concluded tha: the primary factor
> iated with elevated blood lead levels in
> hildren was ingestion or inhalation of
> containing lead. Data on dietary intake
> ad were not obtained because the cli-
> and proximity to the smelter prevent-
> y farming in the area. It was unlikely
> the dietary lead intakes of the children
> near the smelter and farther away
> significantly different. (P. 12-15.)

th regard to the report of Yankel
l. at Kellogg, Idaho, the criteria
ment states:

> e factors influenced, in a statistically
> ficant manner, the probability of a
> developing an excessive blood lead
>
> Concentrations of lead in ambient air
> n³
> Concentration of lead in soil (ppm).
> 'ge (years).
> Cleanliness of the home (subjective
> ation coded 0, 1, and 2, with 2 signify-
> irtiest).
> General classification of the parents'
> ation (dimensionless).
> hough the strongest correlation found
> between blood lead levels and air lead
> the authors concluded that it was un-
> ' that inhalation of contaminated air
> could explain the elevated blood lead
> observed. (P. 12-16.)

THE APPROPRIATE RELATIONSHIP
/EEN LEAD IN AIR AND LEAD IN BLOOD

veral commenters questioned the
icy's estimate that, for children,
microgram of lead per cubic meter
μg Pb/m³ results in an increase of two micrograms lead per deciliter blood (μg Pb/dl).

AGENCY RESPONSE

EPA has reviewed the studies discussed in the criteria document which report changes in blood lead levels with different air lead levels. The Agency believes that one of the strongest epidemiological studies is that by Azar et al. in which personal dosimeters were used to measure lead intake. This eliminated some of the uncertainty about the extent to which air quality observations accurately reflect actual exposure. From the Azar data, the relationship of lead in the air to lead in the blood, evaluated at 1.5 μg Pb/m³ was 1:1.8. The Azar study was, however, limited to an adult population.

A clinical study of adults, Griffin et al., gives roughly the same conclusion for a group of adults confined to a chamber with controlled exposure to lead aerosol. This study was conducted over a three month period with control over lead ingestion. As air lead levels in the chamber were increased from 0.15 μg Pb/m³ to 3.2 μg Pb/m³, the air lead to blood lead relationship was 1:1.7.

Because children are known to have greater net absorption and retention of lead than adults, it is reasonable to assume that the air lead to blood lead relationship for this sensitive population, exposed to air lead levels in the range of the proposed standard, is equal to if not greater than for adults. EPA also notes that the air lead to blood lead relationship is nonlinear and may result in a higher ratio at lower air levels.

In an epidemiological study of children near a smelter, Yankel et al., the response of blood lead to air lead, averaged over the exposure range, was 1.9. EPA believes that these studies as well as others reported in the criteria document, support the criteria document's conclusion that:

> Ratios between blood lead levels and air lead exposures were shown to range generally from 1:1 to 2:1. These were not, however, constant over the range or air lead concentrations encountered. There are suggestive data indicating that the ratios for children are in the upper end of the range and may even be slightly above it. There is also some slight suggestion that the ratios for males are higher than those for females. (P. 12-38.)

THE STATISTICAL FORM AND PERIOD OF THE STANDARD

One commenter expressed the view that, due to the lognormal distribution of measured air lead, a not-to-be-exceeded standard of 1.5 μg/m³, calendar month average, would require sources of air lead to achieve control of their emissions to a geometric monthly mean of 0.41 μg/m³ in order to prevent the occurrence of a violation. Another comment expressed the opinion that, with the normal operation of a 6-day sampling schedule, the number of samples which could be collected in the course of a calendar month would not provide a statistically valid estimate of the actual lead air quality for the period.

Several comments questioned the health basis for the selection of the calendar month averaging period.

EPA RESPONSE

EPA accepts the consensus of comments received on the scientific and technical difficulties presented by the selection of a calendar month averaging period. The Agency believes that the key criterion for the averaging period is the protection of health of the sensitive population. In proposing the 1.5 μg/m³ standard, EPA concluded that this air level as a ceiling would be safe for indefinite exposure of young children. The critical question in the determination of the averaging period is the health significance of possible elevations of air lead above 1.5 μg/m³ which could be sustained without violation of the average of 1.5 μg/m³. In the proposed standard, EPA chose a monthly averaging period on the basis of a study showing an adjustment period of blood lead level with a change of exposure (Griffin et al.). Because of the scientific and technical difficulties of the monthly standard, EPA has reexamined this question and concludes that there is little reason to expect that the slightly greater possibility of elevated air lead levels within the quarterly period is significant for health. This conclusion is based on the following points:

(1) From actual ambient measurements, the distribution of air lead levels is such that where the quarterly standard is achieved, there is little possibility that there could be sustained periods greatly above the average value.

(2) While it is difficult to relate the extent to which a monitoring network actually represents the exposure situation for young children, it seems likely that where elevated air lead levels do occur, they will be close to point of mobile sources of lead air pollution. Typically, young children will not encounter such levels for the full 24-hour period reported by the monitor.

(3) There is medical evidence indicating that blood lead levels reequilibrate slowly to changes in air exposure. This serves to dampen the impact of a short-term period of exposure to elevated air lead.

(4) Direct exposure to air is only one of several routes of total exposure. This lessens the impact of a change in air lead on blood lead levels.

On balance, the Agency concludes that a requirement for the averaging of air quality data over calendar quarter will improve the validity of air quality data gathered without a significant reduction in the protectiveness of the standard.

THE APPROPRIATE MARGIN OF SAFETY

Several comments received by the Agency criticized the proposed standard for incorporating an excessive margin of safety. This criticism was based either on the view that the critical health effect, impaired heme synthesis, was not of health significance or on the view that EPA had employed conservative estimates of the several factors used in calculating the standard which, when combined, resulted in an excessively stringent standard.

Other comments were received which expressed concern that the standard had little or no margin of safety, particularly for certain subgroups within the general population of young children.

AGENCY RESPONSE

EPA does not agree that the impairment of heme synthesis is a physiological response to lead exposure that is without health significance. While EPA does not find that this impairment is necessarily serious to health at the point at which it first can be detected by the elevation of erythrocyte protoporphyrin, at a threshold in a range of 15-20 μg Pb/dl, the Agency does believe that above blood levels of 30 μg Pb/dl this effect has progressed to the extent that it should be regarded as an adverse health effect.

In determining the final ambient air standard for lead, EPA has used margin of safety considerations principally in establishing a maximum safe blood lead level for individual children at 30 μg Pb/dl and in determining the percentage of children to be placed below this maximum level, about 99.5 percent. Using these factors, results in a target geometric mean population blood lead of 15 μg Pb/dl.

In establishing other factors used in calculating the standard, EPA has used margin of safety in the sense of making careful judgments based on available data, but these judgments have not been at the precautionary extreme of the range of data available to the Agency. In the case of the geometric standard deviation (GSD), studies reviewed in the criteria document showed a range of 1.3 to 1.5. A standard based on a 1.5 GSD would be far more stringent than using 1.3. EPA took the 1.3, however, because of its concern that the total geometric standard deviation contains variation attributable to monitoring and analytical methodology. In estimating the relationship between air lead and blood lead to be 1:2, the Agency used an epidemiological study of children near a smelter, Yankel et al., where response of blood lead to air lead averaged over the exposure range was 1 to 1.9. In adopting 12 μg Pb/dl as the part of blood lead attributable to nonair sources, EPA is concerned that typical levels for this component may be much greater, and that regulatory actions by other public health programs may be necessary to achieve a 12 μg level.

Because of the variability between individuals in a population experiencing a given level of lead exposure, EPA finds it is impossible to provide the same amount of margin of safety for all members in the sensitive population, or to define the margin of safety in the standard as a simple percentage. EPA does believe that the factors it has used in designing the standard provide an adequate margin of safety for a large proportion of the sensitive population. The Agency does not believe that this margin is excessively large or on the other hand that the air standard can protect everyone from elevated blood lead levels.

THE IMPORTANCE OF THE RESPIRABLE FRACTION OF TOTAL AIR LEAD LEVEL

The Agency received a number of comments expressing concern that, because only a fraction of airborne particulate matter is respirable, an air standard based on total air lead is unnecessarily stringent.

AGENCY RESPONSE

EPA agrees that some lead particles are too small or too large to be deposited in the respiratory system. EPA cannot conclude, however, that particles outside of the respirable range do not represent an exposure hazard. A significant component of exposure can be ingestion of materials contaminated by deposition of lead from the air. In addition to the indirect route of ingestion and absorption from the gastrointestinal tract, nonrespirable lead in the environment may, at some point, become respirable through weathering or mechanical action. EPA concludes, therefore, that total airborne lead, both respirable and nonrespirable fractions, should be addressed by the air standard.

THE ECONOMIC IMPACT OF THE PROPOSED STANDARD

A number of commenters were critical of the Agency's economic impact assessment, and argued that the forecast underestimated the severity of the economic impact to certain lead industries.

AGENCY RESPONSE

The comments critical of the draft impact statement did not include data which would allow EPA to confirm the possibility of more severe economic impacts on certain source categories including primary and secondary lead smelters which could have difficulty in limiting emissions sufficiently to assure attaining the standard in their immediate vicinity. Under the Clean Air Act, the primary responsibility for implementing the standard is assigned to the States and each State is required to submit a plan to EPA demonstrating how attainment is to be achieved. The actual economic impacts of implementation are difficult to estimate at this time since, following promulgation, States will have 9 months to develop and submit these plans to EPA. The plans must demonstrate attainment as soon as practicable, but no later than 3 years following the date of plan approval. However, under certain circumstances, States may request up to a 2-year extension of this deadline. Other sections of the Clean Air Act may be used with the Administrator's discretion to grant further extensions of compliance deadlines for impacted industrial facilities.

EPA cannot at this time accurately predict the impact of this standard, but with the timetable in the Act, sees no reason to expect imminent closure of any facility. The Agency is committed to developing accurate data for specific plants in cooperation with the industry and State agencies in order to avoid the imposition of unnecessary controls. EPA's principal concern, however, must be to follow the mandate of the Clean Air Act relating to the protection of the public health.

EPA believes that the economic impact asessment is a reasonable forecast of the economic consequences of implementation of the standard.

THE PROPOSED STATE IMPLEMENTATION PLAN (SIP) REGULATIONS

A summary of comments and the Agency response is included in the preamble to the final regulations published elsewhere in this FEDERAL REGISTER.

THE FEDERAL REFERENCE METHOD FOR MONITORING LEAD AIR QUALITY

A summary of comments and the Agency's disposition is included in the preamble to the final method published elsewhere in this FEDERAL REGISTER.

THE ADMINISTRATIVE PROCEDURES EMPLOYED BY EPA IN THE DEVELOPMENT OF THE PROPOSED STANDARD AND THE PROVISION FOR PUBLIC PARTICIPATION

Two commenters requested that cross examination of witnesses be allowed in the post-proposal public hearing on the proposed standard and implementation regulations. EPA also received a request to postpone the public hearing and to extend the comment period, citing the need to complete ongoing studies.

AGENCY RESPONSE

Both the request for cross-examination and the extension of the comment period were denied by the Agency. With regard to the request for cross-examination, the Agency determined that, in light of the extensive review already conducted, cross-examination was not likely to produce new information or results that would justify such a significant departure from the normal rulemaking process. Also the existence of the normal comment period was sufficient to allow interested members of the public to raise questions concerning the Agency's determinations. Further, due to the extensive review opportunities available at all stages of the regulatory development, an extension of the comment period was not believed to be sufficiently necessary to further delay the schedule for preparation of the final rule.

CLARIFICATION OF ELEMENTS OF THE STANDARD

From reviewing the comments received, EPA wishes to clarify the following points in the presentation of the rationale for the final standard:

(1) EPA is making a distinction between the blood lead level that is the threshold for detection of the biological effect, impaired heme synthesis, and the blood lead level at which this effect has progressed to an extent that it is regarded as adverse to health.

(2) EPA is making a distinction between estimating a maximum safe blood lead level for an individual child, and establishing a population target geometric mean blood lead level for the sensitive population.

(3) EPA is making a distinction between what the contribution to blood lead levels from nonair sources actually may be, and attributing a contribution from nonair sources for the purpose of standard setting.

DERIVATION OF THE NUMERICAL LEVEL OF THE FINAL STANDARD

EPA's objective in setting the level of the standard is to estimate the concentration of lead in the air to which all groups within the general population can be exposed for protracted periods without an unacceptable risk to health.

This estimate is based on EPA's judgment in four key areas:

(1) Determining the "sensitive population" as that group within the general population which has the lowest threshold for adverse effects or greatest potential for exposure. EPA concludes that young children, aged 1 to 5, are the sensitive population.

(2) Determining the safe level of total lead exposure for the sensitive population, indicated by the concentration of lead in the blood. EPA concludes that the maximum safe level of blood lead for an individual child is 30 μg Pb/dl and that population blood lead, measured as the geometric mean, must be 15 μg Pb/dl in order to place 99.5 percent of children in the United States below 30 μg Pb/dl.

(3) Attributing the contribution to blood lead from nonair pollution sources. EPA concludes that 12 μg Pb/dl of population blood lead for children should be atttributed to nonair exposure.

(4) Determining the air lead level which is consistent with maintaining the mean population blood lead level at 15 μg Pb/dl. Taking into account exposure from other sources (12 μg Pb/dl) EPA has designed the standard to limit air contribution after achieving the standard to 3 μg Pb/dl. On the basis of an estimated relationship of air lead to blood lead of 1 to 2, EPA concludes that the ambient air standard should be 1.5 μg Pb/m^3.

Each of these four areas is discussed further in the following sections.

SENSITIVE POPULATION

EPA believes that the health of young children is at particular risk from lead exposure. This is because children have a greater physiological sensitivity to the effects of lead than do adults and may have greater exposure to environmental lead from playing in contaminated areas. Other sensitive populations identified by EPA include those occupationally exposed, and pregnant women and their fetuses. Comments received on the proposed standard did not challenge EPA's position that young children are the most sensitive population for determining the standard. A number of comments did point out that within the general population of children there were subgroups with enhanced risk due to genetic factors, dietary deficiencies, or residence in urban areas. EPA acknowledges the higher risk status of such groups but does not have information either in the air quality criteria or in the comments received for estimating a threshold for adverse effects separate from that of all young children. Concern about these high risk subgroups has, however, influenced EPA's determination of the percentage of the population of children (99.5 percent) to be maintained below 30 μg Pb/dl.

EPA continues to be concerned about the possible health risk of lead exposure for pregnant women and their fetuses. The stress of pregnancy may place pregnant women in a state more susceptible to the effects of lead, and transplacental transfer of lead may affect the prenatal development of the child. There is, however, insufficient scientific information for EPA to either confirm or dismiss this suggestion, or to establish that pregnant women and fetuses are more at risk than young children.

THE MAXIMUM SAFE EXPOSURE FOR CHILDREN

In determining the maximum safe exposure to lead for children, EPA has taken the measurement of blood lead as the indicator of total lead dose. There are other possible indicators of exposure, for example the level of zinc protoporphyrin (ZPP), but most health studies reported in the criteria document utilize blood lead levels as indications of the mobile body burden of lead. The criteria document reports the following table of effect thresholds for children with increasing blood lead levels.

SUMMARY OF LOWEST OBSERVED EFFECT LEVELS IN YOUNG CHILDREN

	μg Pb/dl
δ-ALAD inhibition	10
Erythrocyte protoporphyrin elevation	15-20
Increased urinary δ-ALA excretion	40
Anemia	40
Coproporphyrin elevation	40
Cognitive (CNS) deficits	50-60
Peripheral neuropathies	50-60
Encephalopathic symptoms	80-100

(P. 13-8.)

The first physiological effect associated with increasing blood lead levels is the inhibition of the enzyme δ-aminolevulinic acid dehydratase (δ-ALAD), both in red blood cells (erythrocytes), and in cells in other tissues. This enzyme catalyzes the condensation of two molecules of δ-aminolevulinic acid (δ-ALA) to form porphobilinogen, one of the components involved in the cellular synthesis of heme. The criteria document reports that the threshold for δ-ALAD inhibition in children is 10 μg Pb/dl.

At blood lead levels above 10 μg Pb/dl, the function of δ-ALAD is increasingly inhibited by lead. The criteria document states that 40 μg Pb/dl is the threshold for elevation of δ-ALA recognized as δ-ALA in the urine or δ-ALA-U, an indication that δ-ALA has begun to accumulate in cells.

EPA does not regard the inhibition of δ-ALAD above 10 μg Pb/dl as adverse to health because of the absence of evidence that there is an impairment of heme synthesis until a threshold of 40 μg Pb/dl is reached. The accumulation of δ-ALA above normal levels, indicated by δ-ALA-U, is regarded as adverse to health, both because of impaired heme synthesis, and

ossibility that δ-ALA accumula-
itself toxic to cells.

criteria document reports that
a threshold of 15-20 µg Pb/dl
is an elevation of protopor-
in erythrocytes. Protopor-
is an organic chemical com-
used by all cells in the produc-
f heme. In the final stage of
synthesis, erythrocyte proto-
rin (EP) and iron are brought
er in the cell mitochondria. In
resence of lead, this step is
d, possibly by inhibition of the
e ferrochelatase or by interfer-
n the transport of iron across
tochondrial membrane. Without
oration into heme, the levels of
orphyrin in the cell become ele-

review of the information pro-
by the air quality criteria docu-
s well as the evidence and argu-
offered by medical professionals
nting on the proposed stand-
'A has concluded that the ef-
f lead on the cellular syntheis
ne, as indicated by elevated
cyte protoporphyrin, are po-
y adverse to the health of
children. This appears, howev-
be a question of the degree to
the effect has progressed. EPA
ot believe that there is signifi-
sk to health at the point where
vation of EP can first be corre-
vith an increase in blood lead
20 µg Pb/dl). On the other
EPA regards as clearly adverse
lth the impairment of heme
sis, and other effects of lead
result in clinical symptoms of
above 40 µg Pb/dl. These ef-
re followed quickly by the risk
vous system deficits for some
n with blood lead levels of 50 µg

has concluded that the maxi-
afe blood lead level for an indi-
child is 30 µg Pb/dl. This is
n the following factors:
he maximum safe blood lead
hould be somewhat lower than
eshold for a decline in hemo-
evels (40 µg Pb/dl).
he maximum safe blood lead
ould be at an even greater dis-
elow the threshold for risks of
system deficits (50 µg Pb/dl).
he maximum safe blood lead
hould be no higher than the
lead range characterized as
xposure by the Center for Dis-
ontrol of the Public Health
as endorsed by the American
y of Pediatrics, because of ele-
of erythrocyte protoporphyrin
30 µg Pb/dl).
he maximum safe blood lead
r an individual need not be as

low as the detection point for the initial elevation of EP (15-20 µg Pb/dl).

The criteria document points out that data from epidemiological studies show that the log values of measured individual blood lead values in a uniformly exposed population are normally distributed with a geometric standard deviation (GSD) of 1.3 to 1.5. Using standard statistical techniques, it is possible to use the geometric standard deviation to calculate the mean population blood lead level which would place a given percentage of the population below the level of an effects threshold. A GSD of 1.5 would result in a lower geometric mean, and a more stringent standard. However, because some of the variability in the GSD is from measurement systems, EPA has used a GSD of 1.3.

Recently, analysis of the data collected by New York City's Bureau of Lead Poisoning has shown that populations of children in the New York area consistently have distributions of blood lead values with a GSD of 1.4 to 1.5. With a geometric mean of 15.0 µg Pb/dl, a GSD of 1.4 results in about two percent of the population over levels of 30 µg Pb/dl. A GSD of 1.5 would place more than four percent over 30 µg Pb/dl. EPA is concerned that such results may imply that the standard is not as precautionary as it would be if the actual GSD was 1.3. However, the Agency's best estimate is that some of the GSD is from analytical and monitoring variance, and for this reason, EPA is using the 1.3 value in calculating the final standard.

In EPA's view, use of the 99.5 percent range is not excessive. From 1970 statistics, there are approximately 20 million children in the United States below the age of 5 years, 12 million in urban areas, and 5 million in center cities where lead exposure may be high. Again, knowledge that there are special high risk groups of children within the general population deters EPA from considering lower percentages.

CONTRIBUTION TO TOTAL LEAD EXPOSURE FROM NONAIR SOURCES

In the proposed standard, EPA argued that the air standard should take into account the contribution to blood lead levels from lead sources unrelated to air pollution. No comments were received challenging this argument. EPA continues to base its calculation of the ambient air standard on the assumptions that, to an extent, the lead contribution to blood lead from nonair sources should be subtracted from the estimate of safe mean population blood lead. Without this subtraction, the combined exposure to lead from air and nonair sources would result in a blood lead concentration exceeding the safe level.

EPA notes that the level of the standard is strongly influenced by judgments about nonair contribution to total exposure, and that there are difficulties in attempting to estimate exposure from various lead sources. Studies reviewed in the criteria document do not provide detailed or widespread information about the relative contribution of various sources to children's blood lead levels. Estimates can only be made by inference from other empirical or theoretical studies, usually involving adults. Also, it can be expected that the contribution to blood lead levels from nonair sources can very widely, is probably not in constant proportion to air lead contribution, and in some cases may alone exceed the target mean population blood lead level.

In spite of these difficulties, EPA has attempted to assess available information in order to estimate the general contribution to population blood lead levels from air and nonair sources. This has been done with evaluation of evidence from general epidemiological studies, studies showing decline of blood lead levels with decrease in air lead, studies of blood lead levels in areas with low air lead levels, and isotopic tracing studies.

Studies reviewed by the criteria document show that the geometric mean blood lead levels for populations of children are frequently above 15 µg Pb/dl. In studies reported, the range of mean population blood lead levels for children was from 16.5 µg Pb/dl to 46.4 µg Pb/dl with most studies showing mean levels greater than 25 µg Pb/dl (Fine, 1972; Landrigan, 1975, von Lindern, 1975). EPA believes that, for many of these populations, the contribution to blood lead levels from nonair sources may exceed the desired target mean blood lead level.

In a number of studies, reduction in air lead levels resulted in a decline in children's blood lead levels. A study of blood lead levels in children in New York City showed that children's mean blood lead levels declined from 30.5 µg Pb/dl from 1970 to 1976, while during the same period air lead levels at a single monitoring site fell from 2.0 µg Pb/dl to 0.9 µg/Pb (Billick, 1977). Studies at Omaha, Nebr. (Angle, 1977) and Kellogg, Idaho (Yankel, von Lindern, 1977) also show a drop in mean blood lead levels with declines in air lead levels. As air lead levels decline there appears to be a rough limit to the drop in blood lead levels.

EPA has also examined epidemiological studies in the criteria document where air lead exposure is low, and can be assumed to be a minor contributor to blood lead. These studies provide an indication of blood lead levels resulting from a situation where nonair sources of lead are predominant.

STUDIES REPORTING BLOOD LEAD LEVELS IN CHILDREN EXPOSED TO MODERATE TO LOW AIR LEAD LEVELS

Investigator	Blood lead[1]	Air lead[2]	Comment
Hammer, 1972	11.6	0.1	Children in Helena, Mont.
Angle, 1974	14.4	0.14	Suburban Children ages 1 to 4 in Omaha, Nebr.
Goldsmith, 1974	13.7	0.2 to 0.7 0.3 to 0.8 Children in Crocket, Calif.	Children in Benecia, Calif.
Johnson, Tillery, 1975	10.2	0.6	Female children—mean age 9 in Lakeport, Calif.

[1] In micrograms of lead per deciliter.
[2] In micrograms of lead per cubic meter.

The range of mean blood lead levels in those studies is from 10.2 μg Pb/dl to 14.4 μg Pb/dl, with an average at 12.7 μg Pb/dl.

In addition to epidemiological investigations, EPA has reviewed studies that examine the source of blood lead by detecting characteristic lead isotopes. A study using isotopic tracing (Manton, 1977) suggests that for several adults in Houston, Tex., 7 to 41 percent of blood lead could be attributed to air lead sources. An earlier isotopic study (Rabinowitz, 1974) concluded that for two adult male subjects studied, approximately one-third of total daily intake of lead could be attributed to exposure to air lead levels of 1-2 μg Pb/m³. While these results cannot be directly related to children, it is reasonable to assume that children may exhibit the same or higher percentages of air lead contribution to blood lead level because of a greater potential for exposure to indirect air sources, soil and dust.

From reviewing these areas of evidence, EPA concludes that:

1. In studies showing mean blood lead levels above 15 μg Pb/dl, it is probable that both air and nonair sources of lead contribute significantly to blood lead with the possibility that contributions from nonair sources exceed 15 μg Pb/dl.

2. Studies showing a sustained drop in air lead levels show a corresponding drop in blood lead levels, down to an apparent limit in the range of 10.2 to 14.4 μg Pb/dl.

3. Isotopic tracing studies show air contribution to blood lead to be 7-41 percent in one study and about 33 percent in another study.

In considering this evidence, EPA notes that if, from the isotopic studies, approximately two-thirds of blood lead is typically derived from nonair sources, a mean blood lead target of 15 μg Pb/dl would attribute 10 μg Pb/dl to non-air sources. On the other hand, the average blood lead level from the limited studies available where air exposure was low is 12.7 μg Pb/dl. In the absence of more precise information, EPA is calculating the lead standard based on the attribution of 12 μg Pb/dl of the blood lead level in children to lead sources unaffected by the lead air quality standard. EPA is aware that actual population blood lead levels, either individually or as a population mean, may exceed this benchmark. However, if EPA were to use a larger estimate of non-air contribution to blood lead, the result would be an exceptionally stringent standard, which would not address the principal source of lead exposure.

THE RELATIONSHIP BETWEEN AIR LEAD EXPOSURE AND RESULTING BLOOD LEAD LEVEL

EPA has reviewed the studies discussed in the criteria document which report changes in blood lead levels with different air lead levels. The Agency believes that one of the strongest epidemiological studies is that by Azar et al., which used personal dosimeters to measure lead intake. This eliminated some of the uncertainty about the extent to which air quality observations accurately reflect actual exposure. From the Azar data, the relationship of lead in the air to lead in the blood, evaluated at 1.5 μg Pb/m³, was 1:1.8. The Azar study was, however, limited to an adult population.

A clinical study of adults, Griffin et al., gives roughly the same conclusion for a group of adults confined to a chamber with controlled exposure to lead aerosol. This study was conducted over a three month period with control over lead ingestion. As air lead levels in the chamber were increased from 0.15 μg Pb/m³ to 3.2 μg Pb/m³, the air lead to blood lead relationship was 1:1.7.

Because children are known to have greater net absorption and retention of lead than adults, it is reasonable to assume that the air lead to blood lead relationship for this sensitive population, exposed to air lead levels in the range of the proposed standard, is equal to if not greater than for adults. EPA also notes that the air lead to blood lead relationship is nonlinear which will result in a higher ratio at lower air levels.

In an epidemiological study of children near a smelter, Yankel et al., the response of blood lead to air lead, averaged over the exposure range, was 1.95. This study provided information on the relationship of blood lead to air lead over a very large range of air lead values. The air lead values in the study are the result of a model calibrated by monitoring data. The relative error of the individual slopes, especially in the low range is larger than in the Azar study.

The authors of the study, Yankel and von Lindern, chose a log-linear model which provided a good fit to the data and gave an estimated slope of about 1.2 at an air lead of 1.5. However, EPA sees a problem with a log-linear model in that it forces a lower slope at low air lead values, and a higher slope at higher lead values. This is in direct contradiction to the Azar and the Griffin studies, both of which indicate higher slopes at lower air lead values.

Because of the uncertainties in the low air lead values in the Idaho study, EPA felt that the calculation of an average slope or ratio over the entire range of data would be a moderate compromise. The calculation of an average slope gives a value of 1.95. EPA believes that these studies as well as others reported in the criteria document support the document's conclusion that:

> ratios between blood lead levels and air lead exposures were shown to range generally from 1:1 to 2:1. These were not, however, constant over the range of air lead concentrations encountered. There are suggestive data indicating that the ratios for children are in the upper end of the range and may even be slightly above it. There is also some slight suggestion that the ratios for males are higher than those for females (pp. 12-38.)

CALCULATION OF THE AIR STANDARD

EPA has calculated the standard based on the conclusions reached in the previous sections;

1. Sensitive population: Children, ages 1-5.
2. Health basis: Maximum safe blood lead level for individual children is 30 μg Pb/dl based on concern for impaired heme synthesis above 30 μg Pb/dl and margin of safety for anemia above 40 μg Pb/dl and nervous system deficits above 50 μg Pb/dl.
3. Maximum safe geometric mean blood lead for children based on placing 99.5 percent of the sensitive population below the 30 μg Pb/dl level of concern: 15 μg Pb/dl.
4. Estimate of blood lead level attributed to non-air sources: 12 μg Pb/dl.
5. Allowable contribution to blood lead from air sources after achieving the standard: 15 μg Pb/dl-12 μg Pb/dl = 3 μg Pb/dl
6. Air lead concentration consistent with blood lead contribution from air sources: 3 μg Pb/dl × 1 μg Pb/m³ air/2 μg Pb/dl blood = 1.5 μg Pb/m³.

SELECTION OF THE AVERAGING PERIOD FOR THE STANDARD

Based on comments received and consideration by the Agency, the proposed averaging period of a calendar month is extended to a calendar quarter. EPA believes that this change will significantly improve the validity of lead air quality data which will be gathered to monitor progress toward attainment without placing an undue burden of State and local environmental agencies, or significantly reducing the protectiveness of the standard.

The Agency believes that the key criteria for the averaging period is the protection of the health of the sensitive population. In proposing the 1.5 µg Pb/m³ standard, EPA concluded that this air level was safe for young children with an indefinie exposure period. The critical factor in the determination of the averaging period is the health significance of possible elevations of air lead above 1.5 µg Pb/m³ which could be encountered for short periods without raising average levels to exceed the standard. In the proposed standard, EPA chose a monthly averaging period on the basis of a study (Griffin et al.) showing an equilibration period of blood lead level with a change in exposure. Because of the scientific and technical difficulties of the monthly standard, EPA has reexamined this question and concluded that there is little reason to expect that the slightly greater possibility of elevated air lead levels sustainable by the calendar quarter standard is significant for health. This conclusion is based on the following factors:

(1) From actual ambient measurements, there is evidence that the distribution of air lead levels is such that if the quarterly average was achieved there is little possibility that there could be sustained periods greatly above the average value.

(2) While it is difficult to relate the extent to which a monitoring network actually represents the exposure situation for young children, it seems likely that where elevated air lead levels do occur, they will be close to point or mobile sources. Typically, young children will not encounter such levels for the full 24-hour period reported by the monitor.

(3) There is medical evidence indicating that blood lead levels reequilibrate slowly to changes in air exposure. This serves to dampen the impact of a short-term period of exposure to elevated air lead.

(4) Direct exposure to air is only one of several routes of total exposure. This lessens the impact of a change in air lead on blood lead levels.

On balance, the Agency concludes that a requirement for the averaging of air quality data over a calendar quarter will improve the validity of air quality data gathered without a significant reduction in the protectiveness of the standard.

MARGIN OF SAFETY

The Clean Air Act instructs EPA to set the level of an ambient air quality standard at a level which protects the public health with a margin of safety. One approach to using margin of safety is to estimate the air concentration of a pollutant that is the threshold for the first adverse effect detected with increasing air levels, and then set the air standard at a somewhat lower level. The extent of the safety margin between the standard and the estimated threshold for adverse effects is influenced by such factors as the severity or irreversibility of effects, the degree of uncertainty about known or suspected health effects, the size of the population at risk, and possible interactions of several pollutants in potentiating health effects. While the margin of safety is based on available scientific information, this factor is judgmental in that the Administrator must weigh the acceptability of estimated risk.

Estimating an appropriate margin of safety for the air lead standard is complicated by the multiple sources and media for lead exposure. Because of this, EPA has elected to use margin of safety considerations in estimating the maximum safe level for blood lead, and the percentage of the sensitive population to be placed below this level, rather than making a final adjustment to concentration of lead in the air. EPA has adopted 30 µg Pb/dl as the maximum safe blood lead level for individual children, and the air standard is calculated to maintain most children below this target. On the basis of information developed in the criteria document and from public comment, blood lead levels between 30 and 40 µg Pb/dl are associated with impairments of the heme synthetic pathway which EPA regards as adverse to health. Blood lead levels above 40 µg Pb/dl are associated with a decline in hemoglobin levels, and levels above 50 µg Pb/dl are associated with the risk of nervous system deficits for some children. With a geometric mean population blood of 15 µg Pb/dl lead, most children will be well below these thresholds, but a small percentage can be expected to have blood lead levels of concern.

Because of the variability between individuals in a population experiencing a given level of lead exposure, EPA finds that it is not possible to provide the same amount of margin of safety for all members in the sensitive population, or to define a margin of safety in this standard as a simple percentage. In developing the numerical level of the standard, EPA used evidence in the criteria document that the blood lead levels for individuals in a given population of children are log-normally distributed. The statistical properties of this distribution make it possible to calculate the percentage of the population which will fall below any given blood lead level. Individuals at each of these levels would have a different margin of safety below the maximum safe blood lead level. As a rough example, with a population of children with a geometric mean blood lead of 15 µg Pb/dl, 86 percent of the children would be below 20 µg Pb/dl, 97.5 percent would be below 25 µg Pb/dl and 99.5 percent would be below 30 µg Pb/dl. Assuming a population of children in central urban areas where air lead was at the standard level, 693,000 children would be over 20 µg Pb/dl, 126,500 over 25 µg Pb/dl, and 20,605 above 30 µg Pb/dl.

In determining the appropriate margin of safety, the Agency has also included consideration of the following factors:

(1) In addition to the health effects discussed in the Air Quality Criteria for Lead report, multiple [illegible] involvements of lead in practically all cell types, tissues, and organ systems. The significance for health of these has not been fully studied.

(2) There are no beneficial effects of lead at current environmental levels.

(3) EPA has incomplete data about the extent to which children are indirectly exposed to lead from air lead which moves to other environmental media, such as water, soil and dirt, and food.

(4) Lead is chemically persistent and with continued uncontrolled emissions will continue to accumulate both in human tissue and in the environment.

(5) There is a possibility that lead exposure resulting in blood lead levels previously considered safe may in fact influence the neurological development and learning abilities of the young child. EPA does not have evidence, however, that provides more than a suggestion that this could occur at blood lead levels below 30 Pb/dl for individual children.

IMPACT OF LEAD DUSTFALL ON BLOOD LEAD

In the preamble for the proposed air standard for lead, EPA pointed out that the significance of dust and soil lead as indirect routes of exposure has been of particular concern in the case of young children. Play habits and mouthing behavior between the ages of 1 and 5 have led to the conclusion that greater potential may exist in these children for ingestion and inhalation of the lead available in contaminated dust and soil. EPA is also concerned that the deposition of lead particles can lead to general contamination of the environment and increased lead exposure from surface waters and foodstuffs.

Studies reviewed in the criteria document indicate a correlation between soil and dust levels and childrens' blood lead levels in highly contaminated environments (Yankel and von Lindern, 1977; Barltrop, 1974; Galke, in press). The lead threshold for concern has been reported as 1,000 parts per million (ppm) in soil (Yankel and von Lindern, 1977). At levels of between 500 and 1,000 ppm in soil, the criteria document concludes that blood lead levels begin to increase. A twofold increase in soil concentration in this range is predicted to result in a 3-6 percent rise in blood lead levels. Below 500 ppm lead in soil, no correlation has been observed with blood lead levels.

The normal background for lead in soil is cited in the criteria document as 15 ppm. Due to human activities, the average levels in most areas of the United States are considerably higher. Soil studies conducted by EPA's Office of Pesticides Programs from 1974 to 1976 in 17 urban areas reported only 3 cities with arithmetic mean concentrations in excess of 200 ppm, with the highest value 537 ppm. Concentrations in the soils surrounding large point sources of lead emissions, or heavily travelled roads may reach many thousand ppm.

Because of the many factors involved, EPA is unable to predict the relationship between air lead levels, dustfall rates, and resulting soil accumulation. Complicating factors include: Particle size distribution, rainout, other meteorological factors, topographical features affecting deposition, and removal mechanisms.

EPA believes, however, that significant impacts on blood lead of soil and dust lead are mainly limited to areas of high soil concentration (in excess of 1,000 ppm) around large point sources and heavily travelled roads. Evidence suggests that soil lead levels in areas with air lead levels in the range of the standard are below the threshold for lead health impact (Johnson, Tillery, 1975; Johanson, 1972; EPA, 1975 Air Quality Data and Soil Levels).

Comments received on the proposed standard argued that the lead air standard should be limited to respirable size lead particulate matter, as larger particles would fall to the ground without being deposited or absorbed in the lung. EPA has decided not to accept this recommendation because, as discussed above, larger particles can contribute to lead dose by human ingestion of airborne particles, by contamination of other environmental media, or by eventual reduction to respirable size by mechanical action or weathering.

WELFARE EFFECTS

Comments received on the proposed lead air quality standard did not address the issue of welfare effects or the need for a secondary air quality standard more restrictive than the primary standard. EPA maintains its position that the primary air quality standard will adequately protect against known and anticipated adverse effects on public welfare. EPA does not have evidence that a more restrictive secondary standard would be justified.

Available evidence cited in the criteria document indicates that animals do not appear to be more susceptible to adverse effects from lead than man, nor do adverse effects in animals occur at lower levels of exposure than comparable effects in humans.

Lead is absorbed but not accumulated to any great extent by plants from soil. Lead is either unavailable to plants or is fixed in the roots and only small amounts are transported to the above ground portions. Lead may be deposited on the leaves of plants and present a hazard to grazing animals. Although some plants may be susceptible to lead in the natural environment, it is generally in a form that is largely nonavailable to them.

There is no evidence to indicate that ambient levels of lead result in significant damage to manmade materials. Effects of lead on visibility and climate are minimal.

Based on such data, EPA promulgates the secondary air quality standard for lead at 1.5 μg Pb/m^3, calendar quarter average.

ECONOMIC IMPACT ASSESSMENT

As required by Executive Orders 11821 and 12044, EPA has conducted a general analysis of the economic impact which might result from the implementation of the lead regulations. This analysis was not intended for nor was it used in the development or promulgation of the standard, and was issued for informational purposes only.

The economic impact assessment points out that the categories of sources likely to be affected by control of lead emissions are primary lead and copper smelters, secondary lead smelters, gray iron foundries, gasoline lead additive manufacturers, and lead storage battery manufacturers. This analysis further indicates that some primary and secondary lead smelters and copper smelters may be severly strained economically in achieving emission reductions that may be required in implementing the proposed air quality standard.

There are, however, uncertainties associated with evaluating the impact of attaining the standard. For smelters and foundries, attaining the standard may require control of fugitive lead emissions, i.e., those emissions escaping from individual process operations, other than emissions from smoke stacks. Fugitive emissions are difficult to estimate, measure, and control; and it is also difficult to predict their impact on air quality near the facility. From the information available to EPA, nonferrous smelters may have great difficulty in achieving lead air quality levels consistent with the proposed standard in areas immediately adjacent to the smelter complex.

The change in averaging time from a monthly average to a calendar quarter average will affect the economic impacts associated with the lead standard because for a given level of the standard, a longer averaging period is theoretically less stringent than a shorter averaging period.

OTHER LEAD REGULATORY AND CONTROL PROGRAMS

EPA's ambient air quality standard is only one of a number of Federal, State, and local programs designed to limit exposure to lead.

In 1975, EPA promulgated the national interim primary drinking water regulation, setting a maximum contaminant level for lead. The standard, aimed at protecting children from undue lead exposure, was set at 50 μg Pb/liter. In 1977, the National Academy of Sciences concluded that a lead level at which adverse health effects are observed cannot be set with assurance at any value greater than 25 μg Pb/liter. The Office of Drinking Water is currently considering the need to revise the interim drinking water standard for lead.

Based on its toxicity, EPA has included lead on its list of priority water pollutants for which effluent guidelines are being developed under the Clean Water Act. Effluent guidelines are being developed for lead for nonferrous smelters, based on achievement of best available technology.

EPA's Office of Pesticide Programs has promulgated regulations based on the toxicity of lead which require the addition of coloring agents to the pesticide lead arsenate and specify disposal procedures for lead pesticides. Use of lead in pesticides is a small and decreasing proportion of total lead consumption in the United States.

The Resource Conservation and Recovery Act (RCRA) of 1976, through which EPA is to establish standards on how to treat, dispose, or store hazardous wastes, provides a means for specifying how used crankcase oil and other waste streams containing lead should be recycled or safely disposed of. Regulatory actions related to wastes containing lead are currently being developed under subtitle C of RCRA.

EPA has regulations for reducing the average lead content in the total gasoline pool to 0.5 grams/gallon by October 1, 1979, and regulations providing for lead-free gasoline required for cars equipped with catalytic converters and other vehicles certified for use of unleaded fuel. The former regulations are based on reducing exposure to airborne lead to protect public health. Other EPA actions which result in the reduction of airborne lead levels include ambient standards and State implementation plans for other pollutants such as particulate matter and sulfur dioxide and new source performance standards limiting emissions of such pollutants. Existing and new sources of particulate matter emissions generally use control techniques which reduce lead emissions as one component of particulate matter.

The Occupational Safety and Health Administration proposed regulations in 1975 to limit occupational exposure to lead to 100 μg Pb/m^3, 8-hour time-weighted average. The exposure limit was based on protecting against effects, clinical or subclinical, and the mild symptoms which may occur below 80 μg Pb/dl, providing an adequate margin of safety. The level of 100 μg Pb/m^3 is anticipated to limit blood lead levels in workers to a mean 40 μg Pb/dl and a maximum of 60 μg Pb/dl. OSHA is presently reviewing the latest information on lead exposure and health effects in preparation for promulgation of the workplace standard for lead.

The Department of Housing and Urban Development (HUD) has requirments for reducing human exposure to lead through the prevention of lead poisoning from ingestion of paint from buildings, especially residential dwelling. Their activities include (1) prohibition of the use of lead-based paints on structures constructed or rehabilitated through Federal funding and on all HUD-associated housing; (2) the elimination of the immediate hazard from lead-based paint; (3) notification of purchases of HUD-associated housing constructed prior to 1950 which may contain lead-based paint; and (4) research activities to develop improved methods of detection and elimination of lead-based paint hazards, and the nature and extent of lead poisoning.

The Consumer Product Safety Commission (CPSC) promulgated regulations in September 1977 which ban: (1) Paint and other surface coating materials containing more than 0.06 percent lead; (2) toys and other articles intended for use by children bearing paint or other similar surface coating material containing more than 0.06 percent lead; and (3) furniture coated with materials containing more than 0.06 percent lead. These regulations are based on CPSC's conclusion that it is in the public interest to reduce the risk of lead poisoning to young children from ingestion of paint and other similar surface-coating materials.

The Food and Drug Administration (FDA) adopted in 1974 a proposed tolerance for lead of 0.3 ppm in evaporated milk and evaporated skim milk. This tolerance is based on maintaining children's blood lead levels below 40 μg Pb/dl. FDA has also proposed an action level of 7 μg Pb/ml for leachable lead in pottery and enamelware, although the exact contribution of such exposure to total human dietary intake has not been established.

The Center for Disease Control (CDC) concluded in 1975 that undue or increased lead absorption exists when a child has confirmed blood lead levels of 30-70 μg Pb/dl or an EP elevation of 60-189 μg Pb/dl except where the elevated EP level is caused by iron deficiency.

In developing the lead air standard, EPA has estimated both individual and population blood lead levels which it regards as safe targets. The Agency believes that these targets do not necessarily serve as precedents for other regulatory programs. There are three reasons for this view:

(1) These targets were selected on the basis of what the Clean Air Act requires. Other programs have other legislative requirements which would lead to adoption of different but equally legitimate goals.

(2) The scientific data provided by the air quality criteria allow comparison of air levels with blood lead levels, but analogous information is not available for other media. At this time, there does not appear to be the same extent of information about the impact on blood lead of lead in food, water, and nonfood ingested items. Because of this, FDA, CPSC and other EPA standards have been based on estimates of acceptable daily dose rather than on blood lead targets.

(3) Studies currently underway may provide new information relevant to estimating safe levels of lead exposure.

COMMENTS BY OTHER FEDERAL AGENCIES

Comments on the proposed lead air quality standard were received from eight Federal Agencies. Five of the Agencies endorsed the air standard while three of the Agencies commented on specific issues and neither endorsed nor opposed the standard. The Center for Disease Control and the U.S. Public Health Service voiced support for the proposed standard of 1.5 μg Pb/m^3 and urged basing the decision on the standard solely on considerations of public health. CDC is fully satisfied that EP elevation does indeed represent a subclinical manifestation of lead toxicity and that young children are the population most at risk from lead exposure, while some subgroups of children are at special risk to lead because of conditions such as malnutrition, genetic factors, or iron deficiency.

The Consumer Product Safety Commission endorsed the approach and some of the judgments made in arriving at the proposed air standard. CPSC concurred with the position that children are the population at enhanced risk to lead exposure, and that the goal of a mean population blood lead level for children of 15 μg Pb/dl is sufficiently low to be protective of the population at enhanced risk of exposure. CPSC views the selection of EP elevation as the adverse health effect of concern as open to challenge and suggests basing the standard on a more generally recognized severe health effect. CPSC concurs that the contribution of nonair sources to lead body burden must be evaluated in setting the air standard and suggests that a larger nonair contribution, such as 13.5 μg Pb/dl used in the California standard, might be considered.

The Food and Drug Administration commended EPA's proposal of an ambient air quality standard for lead. FDA agrees that children aged 1-5 years old comprise the most critically sensitive population. FDA concurs that 15 μg Pb/dl is a reasonable maximum blood lead level to use as an average national goal for children aged 1 to 5, although FDA suggests that for young children the margin of safety is disturbingly narrow. The division of the 15 μg Pb/dl into 12 μg Pb/dl for nonair sources and 3 μg Pb/dl for air sources was not unreasonable in FDA's view.

The Occupational Safety and Health Administration endorsed EPA's proposed standard for lead and agrees with EPA that 15 μg Pb/dl as an average national blood lead level goal for young children is reasonable. OSHA views their proposed standard of 100 μg Pb/m^3, 8-hour time-weighted average, and their establishment of 40 μg Pb/dl as the threshold effect level for workers as consistent with the EPA proposed standard.

The Department of Transportation (DOT) endorsed the proposed standard of 1.5 μg Pb/m^3. Based on an analysis of the impact of the proposed standard on the highway program, DOT concluded that it is highly probable that transportation-related violations of the proposed standard would be limited to large urban areas.

In commenting on the proposed standard, the Department of the Interior (DOI) expressed concern that the burden for meeting the proposed standard will fall primarily on lead and copper smelters and battery manufacturers, and commented on the impact of lead dustfall on ground water quality. The Tennessee Valley Authority provided specific comments on the proposed State implementation plan regulations and the proposed Federal reference method. The Department of Commerce offered comments on the potential impacts of the standard, pointing out that more consideration should be given to the potential impact of the standard on the petroleum industry.

THE FEDERAL REFERENCE METHOD

The reference method for the determination of lead in suspended particulate matter collected from ambient air describes the appropriate techniques for determining the concentration of lead and its compounds as measured as elemental lead in the ambient air. A total of eight organizations submitted written comments on the method and two persons made comments at EPA's February public hearing on the proposed air quality standard. Since proposal of the Federal reference method for lead, EPA has completed additional testing of the method and added new information on the precision of the extraction analysis procedure.

Two of the commenters recommended the addition of a nitric plus hydrochloric acid extraction procedure. The extraction procedure of the proposed method contains only nitric acid. Use of a mixed acid procedure would permit the analyst to quantitatively extract more metals than just lead, thereby allowing him to analyze the same extract for more than one metal. The analysis for lead would not be affected. EPA agrees that a mixed acid extraction procedure should be added, and the revised method contains a mixed nitric-hydrochloric acid extraction procedure.

One commenter questioned the reliability of the air volume measured in the sampling procedure because of differences between initial and final flow rates caused by buildup of particulate matter on the collecting filter. The method of sampling specifies that initial and final flow rates must fall between 40 and 60 cubic feet per minute and variations within this range cause only a slight error. If the flow rate specification is not met, the sample should be voided. For these reasons, EPA believes the air volume measurement does not suffer unduly from inaccuracies.

A question was raised as to the effect of variation in lead content across the filter of the collected sample on lead analysis, since the method calls for analysis of only one strip or one-twelfth of the filter. Our work has shown that strips taken from different positions within the filter can, on occasion, produce different lead values, but the effect appears to be significant only when sampling near a heavily traveled roadway. The proposed method recommends analyzing additional strips, when sampling near a roadway, to minimize this error.

One commenter pointed out that the proposed sampling procedure does not collect gaseous (organic) lead compounds and recommended that EPA consider requiring the use of a method for monitoring gaseous lead. As the criteria document states, reported ambient levels of gaseous lead are very low and EPA has determined that the effort required to carry out the difficult task of monitoring for ambient gaseous lead is not justified in view of the extremely low concentration.

It was pointed out in the preamble to the proposed method that other analytical principles would probably be handled by provision for approval of the equivalent methods (40 CFR Part 53) proposed elsewhere in this FEDERAL REGISTER. Two organizations submitted requests that alternate methods (X-ray fluorescence and anodic stripping voltametry) for lead analysis be declared equivalent to the reference method. These requests will be considered when the procedures for determining equivalency are promulgated.

The final Federal reference method is based on measuring the lead content of suspended particulate matter on glass fiber filters using high volume sampling. The lead is then extracted from the particulate matter with nitric acid facilitated by heat or by a mixture of nitric acid and hydrochloric acid facilitated by ultrasonication. Finally, the lead content is measured by atomic absorption spectrometry.

The reference method specified for lead measures the lead for a single sampling period by extraction of a portion of a high-volume glass fiber filter used to collect particulate matter over a 24-hour period. Some agencies may prefer to composite filter strips from a number of sampling periods and extract and analyze it for lead. This procedure is acceptable provided the Agency shows that the compositing procedure results in the same average lead value as would be obtained from averaging individual values.

Dated: September 29, 1978.

DOUGLAS M. COSTLE,
Administrator.

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40 CFR Part 50 is amended by adding a new § 50.12 and a new appendix G as follows:

§ 50.12 National primary and secondary ambient air quality standards for lead.

National primary and secondary ambient air quality standards for lead and its compounds, measured as elemental lead by a reference method based on appendix G to this part, or by an equivalent method, are: 1.5 micrograms per cubic meter, maximum arithmetic mean averaged over a calendar quarter.

(Secs. 109, 301(a) Clean Air Act as amended (42 U.S.C. 7409, 7601(a)).)

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APPENDIX G—REFERENCE METHOD FOR THE DETERMINATION OF LEAD IN SUSPENDED PARTICULATE MATTER COLLECTED FROM AMBIENT AIR

1. *Principle and applicability.*

1.1 Ambient air suspended particulate matter is collected on a glass-fiber filter for 24 hours using a high volume air sampler.

1.2 Lead in the particulate matter is solubilized by extraction with nitric acid (HNO_3), facilitated by heat or by a mixture

II. Biochemical/Nervous System Effects
At Levels Below 30 ug/dl

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Medical Sciences

Threshold for lead damage to heme synthesis in urban children

(erythrocyte protoporphyrin/environmental health/ferrochelatase)

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ABSTRACT Although lead has no physiological function and is present in only negligible amounts in the blood of remote populations, it has become customary to accept the usual blood Pb level(s) (BPb) observed in industrialized society as "normal." Pb interferes with many biochemical systems, among them the heme biosynthetic pathway; this is reflected by an exponential increase in erythrocyte protoporphyrin concentration (EP) as BPb increases. The present study estimated the threshold BPb at which an increase of EP occurs in a population of urban children. In the 2,004 children studied, BPbs ranged from 2 to 98 μg/dl, with 1,852 having a BPb of ≤30 μg/dl, a value presently considered normal. Preliminary analysis suggested that an exponential increase in the concentration of EP occurred after a threshold BPb (apparently between 12 and 20 μg/dl) was reached. Precise definition of the threshold BPb for an increase of EP was next determined by two approaches: segmented line techniques and probit analysis. Whether the entire population was analyzed or only the subset of samples with "normal" BPb (≤30 μg/dl), both methods yielded a threshold BPb of 15–18 μg/dl (average value, 15.5). These studies indicate that the heme synthetic pathway is affected by Pb at a level of exposure commonly observed in urban children, which is well below the limit that is presently too easily accepted as normal.

Lead is a nonessential element to the human body. However, accumulation of excessive amounts in the human body is an unavoidable hazard of living in our present day environment (1). There is a gradient in the body burden of Pb [expressed by the blood Pb level (BPb)] from the nearly negligible values observed in remote populations (2, 3) to the highest levels observed in urban dwellers (4). Urban children, who are exposed to high concentrations of Pb in dust from vehicular emission and often from Pb-containing paint in deteriorated housing, tend to have the highest BPb among nonindustrially exposed populations (4). High doses of Pb in children result in severe neurological toxicity, leading to death in the most severe cases (5); exposure to lower doses results in more subtle damage (6). Because Pb is a poison of the SH groups and has affinity for several intracellular structures, it exerts its damage by interference with essential enzymes and cellular functions (7). Among the biochemical pathways that are damaged by Pb, the process of heme synthesis stands prominent because of its physiological importance to all tissues. In view of our detailed knowledge of this pathway, of the availability of sensitive techniques for its study, and of the ease of sampling blood, damage caused to it by Pb can be precisely quantitated. Fluorometric techniques allow precise measurements of the erythrocyte protoporphyrin concentration (EP) (8). This is the substrate of the last step of heme synthesis (i.e., the insertion of iron into the completed protoporphyrin molecule), which takes place in the mitochondria and is catalyzed by the enzyme ferrochelatase, located in the inner cristae. EP increases exponentially with BPb (9), reflecting in the peripheral blood the interference by Pb on mitochondrial function in the erythroid precursors in the bone marrow and in all other tissues.

Previous studies of the effect of Pb on EP have focused on individuals, both urban children (9–11) and adult Pb workers (12, 13), with BPb clearly increased above the usual range. The present study was directed to estimate the threshold BPb for an increase of EP in a population of urban children, of which the great majority had BPbs below the value which is currently called "normal" (14).

MATERIALS AND METHODS

Population Sampled. The experimental sample consisted of 3,830 venous blood specimens obtained in 1976 from children at a variety of locations throughout New York City, collected in Pb-free heparinized tubes, and submitted for Pb analysis to the New York City Department of Health Bureau of Laboratories. Each sample was accompanied by a form that supplied the following information: name, address, birth date, sex, ethnic group, and whether the child had been pretested, hospitalized, or suspected of Pb poisoning or whether the sample was taken for the purpose of screening. In addition, the computerized records of the New York City Bureau of Lead Poisoning Control, where every child found to have a BPb of >40 μg/dl has been listed since 1973, were analyzed. A review indicated that 2,597 of these samples had been obtained exclusively for primary screening for Pb poisoning. The remaining 1,233 samples were excluded for one of the following reasons: duplicate sample, pretested child, suspicion of Pb poisoning, chelation therapy, BPb >40 μg/dl at any previous time, hospitalization for any reason, or incomplete data. The samples were next sorted by age, and an additional 593, obtained from children age <24 months, were also excluded to minimize the frequency of EP increase secondary to iron deficiency (FeD), which is prevalent in this age group (15). The final study group consisted of 2,004 blood samples from children ages 2–12 yr (mean age, 5.5 yr; median age, 4.7 yr), obtained exclusively for primary screening for Pb poisoning from ambulatory children. However, the records of all discarded samples were kept in a separate file to verify whether their inclusion would significantly alter the later findings.

BPb and EP Measurements. BPb was measured by atomic absorption spectrophotometry (16). EP was measured by ex-

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Abbreviations: BPb, blood lead level; EP, erythrocyte protoporphyrin concentration; FeD, iron deficiency; AmLev, δ-aminolevulinic acid; df, degrees of freedom.

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traction and fluorometry (8). Both BPb and EP were measured in μg/dl of blood; hereafter, these units will be omitted. The laboratory that performed the analysis participates successfully in the proficiency testing program of the Center for Disease Control and the New York State Department of Health for both tests.

RESULTS

BPb. In the 2,004 samples in the study group, BPb ranged between 2 and 98, with a distribution that approached log normality. The geometric mean, $\bar{x}$, was 17.3 ($\log_{10} \bar{x} = 1.238 \pm 0.227$).

Correlation Between BPb and EP. EP increased exponentially ($\log_{10}$ EP = 1.099 + 0.016 BPb; correlation coefficient, $r = 0.509$, degrees of freedom (df) = 2,002, $P < 0.001$). In a sample of this size, the existence of correlation, even at this level of significance, does not necessarily imply a relationship between the dependent variable (EP) and the independent one (BPb) that is constant throughout the entire range of the latter. The effect may occur only in the higher range, yet, when the entire group is analyzed as a whole, the predominance of samples in the higher range gives the impression of a significant correlation throughout. Therefore, we analyzed two close, but nonconsecutive, subsets of samples—one with the lowest BPb (2–12) and the other with slightly higher levels (20–30). It became apparent that, in the group with the lowest BPb, there was no correlation with EP ($r = -0.021$; df = 313; $P = 0.71$), whereas, in the group with the higher BPb, there was a significant exponential correlation ($\log_{10}$ EP = 0.923 + 0.021 BPb; $r = 0.254$; df = 634, $P = 0.003$). When all samples with BPbs of >20 were considered, an essentially similar significant correlation persisted ($\log_{10}$ EP = 0.910 + 0.022 BPb; $r = 0.603$, df = 784, $P < 0.001$). [It must be noted that both of these slopes of correlation are essentially the same as in our original study, which had included 1,038 children, 647 of whom had BPbs of >30 (9).] These findings suggested that the exponential increase of EP with BPb does not occur until the latter reaches a given value but thereafter continues at a steady rate, without evidence of a plateau phenomenon, at least in the range of our sample. Thus, a preliminary analysis indicated the existence of a threshold BPb (presumably between 12 and 20) above which EP increases at a constant exponential rate. The further detailed analysis was then directed to achieve a precise definition of this threshold BPb by appropriate techniques.

Estimates of the Threshold BPb for EP from the Entire Population (2,004 Samples). *Segmented line techniques.* The data were analyzed by the segmented curve-fitting technique of Hudson (17), which estimates the intersection (join point in the terminology of refs. 17 and 18) of two regression lines. This has been modified by Hasselblad *et al.* (18) to determine a "hockey stick" regression, specifically to measure threshold levels of biological effect. These two techniques use identical methods to estimate the intersection (threshold level), the Hasselblad technique assumes a horizontal line (no effect) below the threshold level, an assumption probably more valid in biological situations. The intersection was estimated at 17.7 (confidence limits = 16.2 and 21.5) by the Hudson technique and at 18.3 (confidence limits = 17.1 and 20.2) by the Hasselblad technique (Fig. 1A).

Probit analysis. The increase in EP with BPb indicates a dose–response relationship to exposure to Pb because a linear increase in BPb reflects an exponential increase in Pb body burden (7). This relationship can be measured best by the classical technique of probit analysis (19), requiring evaluation of the frequency of an event in groups of individuals exposed to progressively increasing doses. Thus, the data were subdivided into 13 categories: the first category included BPbs 2–10, the next 10 categories spanned the BPbs between 11 and 30 (2 μg/

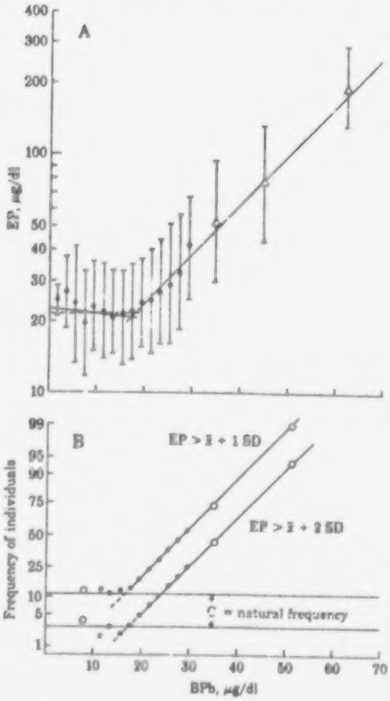

FIG. 1. Determination of the threshold BPb for EP increase with the entire population sample. (*A*) By segmented line techniques. The threshold level was determined from the intersection of two exponential regression lines of EP on BPb. By the method of Hudson (17), the threshold level of 17.7 was estimated from the intersection of the two solid regression lines. The method of Hasselblad *et al.* (18) assumes a horizontal (no effect) line (shown as a dashed line), which intersects the regression line of positive slope identical to that derived above, at a threshold level of 18.3. All 2,004 data pairs were used in these regression analyses. For graphic representation, the data have been grouped on the basis of BPb as follows: ●, 1–30 (15 pairs); and ○, 31–40 (mean = 34.7), 41–50 (mean = 44.8), and 51–98 (mean = 62.7). The vertical bars from each point indicate one SD from the group mean. (*B*) By probit analysis. The data were grouped into 13 categories on the basis of BPb as follows: ●, 11–30 (10 pairs); and ○, 2–10 (mean = 8.1), 31–40 (mean = 34.7), and 41–98 (mean = 51.1). From the lowest BPb group, a "normal" reference EP was determined to be 21.7. For each BPb category, the probit of the frequency of individuals with EPs of >33 and >53 [the geometric mean $\bar{x}$ plus 1 SD (F1) and the mean plus 2 SD (F2) of the reference category, respectively] was plotted against the corresponding BPb. By iterative technique, the ordinate value *C* (representing the "natural" occurrence of the effect) was determined to be 10.7% for F1 and 2.4% for F2. By using only BPb 17–98, the probit regression lines for F1 and F2 were derived. The intersection of these regression lines with their respective ordinate value *C* occurred at BPbs of 16.4 and 16.6, respectively. These values indicate the threshold at which response exceeds the "natural" frequency.

dl each), and the final groups included BPbs 31–40 and 41–98, respectively. Within each category, EP distributions approached log normality, suggesting that any progressive increase with BPb is due to an effect on the entire population and not just on a few more-sensitive individuals. In view of the known effect of Pb on EP, also shown by the data, it is difficult to establish a normal reference group because, in New York City, children with BPb in the range expected in humans living in an uncontaminated environment (2, 3) are extremely rare. Therefore, the lowest BPb category (2–10) was chosen as the most appropriate reference group. This consisted of 131 samples with a geometric mean EP of 21.7 ($\log_{10} \bar{x} = 1.336 \pm 0.198$). For each of the 13 BPb categories, the frequency of individuals with EPs of >33 and >53 (the mean plus 1 SD and plus 2 SD, respectively, of the reference category) were measured and were called F1 and F2, respectively. When the values were transformed into provisional probits and plotted, it became obvious that, at low BPb, the frequencies were rather low but constant and then increased at higher BPb in a linear fashion.

These findings suggested a plateau of no response followed by a dose-dependent response. In this situation the intersection of the probit regression line with the plateau represents the threshold of the dose–response effect. Probit analysis allows a precise estimate of the plateau, the ordinate value *C*, which corresponds to the natural occurrence of the effect in the population. (In this case, *C* reflects the frequency of EP increase due to non-Pb-related causes, such as FeD or other minor chronic diseases.) The values of *C* were determined by iterative probit analysis (19) with all 13 BPb categories. These were 10.7% for F1 and 2.4% for F2. Next, probit regression lines were computed by using *C* set at 0 and only the highest BPb categories. A series of such regression lines was computed by utilizing initially only the seven BPb groups from 40–98 down to 21–22 and then adding into the computation the next lower group, the intersection of each line with the ordinate value *C* was then computed (we shall refer to this as an intercept with the *C* ordinate). As progressively lower BPb groups were added, there was no significant decrease in the values of the intercept until the BPb group 15–16 was included. Thus, the lines derived with the nine BPb groups between 17 and 98 were taken to provide the most correct intercept values. Both these probit regression lines intercepted the *C* ordinate near the abscissa value of 16.5 (16.4 and 16.6, respectively, Fig. 1*B*). For both lines, the observed and expected frequencies were not significantly different ($\chi^2 = 3.337$, df = 7, $P > 0.8$, and $\chi^2 = 3.539$, df = 7, $P > 0.8$, respectively). The intercept values indicate the dose level at which the response starts to exceed the natural frequency (threshold level of the effect). Thus, these data indicate that when the BPb exceeds the value of 16, the frequency of individuals with elevated EP increases in a dose-dependent relationship.

Estimates of the Threshold BPb for EP from the Normal Population of Children (1,852/2,004 Samples). Because a BPb of 30 is the currently accepted upper limit of normal (14), further analysis was then limited to the 1,852 pairs of values from children with BPbs of ≤ 30. Confining the analysis only to this group provides a more accurate estimate of the threshold BPb at which an increase of EP starts in children who would not require medical attention under current standards of care. Additionally, removing from the analysis children with BPbs clearly in the abnormal range avoids any possibility that a more pronounced effect of Pb in the higher range may result in deviation from the linearity of the regression line, which, in turn, may influence the estimation of the intersection.

Segmented line techniques. By using the techniques of segmented line analysis, the intersection was estimated at 15.4 (confidence limits = 12.9 and 18.2) by the Hudson method and at 16.5 (confidence limits = 14.3 and 18.5) by the Hasselblad method.

Probit analysis. Both probit regression lines intercepted the *C* ordinate near the abscissa of 16 (15.9 and 16.4, respectively). Again, for both probit regression lines, the observed and expected frequencies were not significantly different ($\chi^2 = 2.360$, $P > 0.7$, df = 5; $\chi^2 = 2.048$, $P > 0.8$, df = 5).

The threshold estimates obtained by utilizing only the data from children with BPbs in the range presently considered normal appear to be in close agreement with those obtained by using the entire group. Therefore, the previous estimates cannot be ascribed to an artifact.

DISCUSSION

The clinically adverse effects of exposure to large Pb doses are well established (4). It appears logical that there should be a progression from negligible effects at minimal dose, through mild effects at moderate dose, to serious clinical effects at very large dose. However, mild effects are often difficult to document in single individuals. Because Pb is a nonessential element and an extremely toxic one, Patterson postulated in 1965, on the basis of purely geophysical considerations, that the Pb content of the natural man (i.e., man unexposed to the environmental redistribution of Pb caused by human activities) should be negligible (1). Recent studies have clearly shown that, at extremely low exposure, the body Pb content is negligible. Ancient human skeletons have a Pb content several orders of magnitude lower than that in modern bones (20, 21). Even today, the BPbs of remote populations [both "acculturated" (3) and "unacculturated" (2)] are much lower than those of populations living in the industrialized world, with values approaching the low levels predicted by Patterson (1). These studies reaffirm the concept that the Pb body content observed in populations from industrialized countries reflects environmental pollution, therefore, their BPbs as currently observed cannot be considered normal because they are so much higher than in the natural man.

Patterson also predicted that adverse biological effects of Pb would become obvious when searched for with appropriate techniques. This hypothesis also is supported by recent experimental evidence. In 1970, Hernberg and co-workers demonstrated that the activity of the enzyme *d*-aminolevulinic acid (AmLev) dehydratase is inversely proportional to BPb (22). The rate of inhibition is exponential without any apparent threshold, 50% inhibition occurs at a BPb as low as 16.7 (a value typical for most urban dwellers and well within the range presently called normal), and inhibition of 90% occurs at a BPb of 55.5, above which overt clinical symptoms become frequent in children (4). Removal of Pb from the enzyme by means of SH-group reagents results in its complete reactivation (23). The exponential decrease in AmLev dehydratase with an increase in BPb is reflected by an exponential increase in urinary excretion of AmLev (24). The accumulation of EP in severe Pb poisoning has been known for decades (25). The introduction of sensitive fluorometric techniques by our laboratory led to the demonstration that the EP increases exponentially with a linear increase in BPb (8, 9). These observations were confirmed both in children (10, 11) and in adult Pb workers (12, 13). Most of these studies have utilized populations with excessive exposure to Pb, including our initial report that was based on a large number of children, the majority of whom had BPbs of >30 (9). It generally has been assumed that no threshold BPb exists. Recently Roels and coworkers compared two small groups of rural children with different BPbs because only one lived in the proximity of a smelter, and they suggested a lack of correlation with the EP in the group with the lowest BPb (26). However, because there was no overlap in BPbs between the two populations in their studies, an estimate of the threshold level is not possible from their data. The population of children screened in New York City offered, on the other hand, an unusual opportunity for this type of evaluation because it included an adequately large number of individuals, the majority of whom had BPbs within the "normal" range, and its overall BPb distribution was log normal as expected (4). The analysis of the relationship between BPb and EP confirmed the exponential correlation. However, a preliminary analysis suggested that this may not be present throughout the entire range. The data were then analyzed with techniques appropriate to measure threshold levels: (*i*) the segmented line techniques, which estimate the threshold as the intersection of two regression lines, and (*ii*) the probit analysis, which yields a threshold at the intersection between the slope of the dose–response relationship and the line of natural frequency of the dose-unrelated effect. The analysis was first performed with all data, including 1,852 samples with BPbs of ≤ 30 (within the so-called normal range) and 152 with BPbs of >30. The segmented line techniques estimated the threshold at 17.7 by the method of Hudson and at 18.3 by the Hasselblad modification; the probit analysis estimated the threshold at 16.4 and 16.6. Next, the analysis was repeated with only those 1,852/2,004 samples with BPbs of ≤ 30. The restriction of the analysis to this group was motivated by the desire to assess the threshold level in that segment of the population that had BPbs certainly within the "normal" range. The segmented line techniques estimated the threshold at 13.4 by the method of Hudson and at 16.5 by the Hasselblad modification. The probit analysis estimated the thresholds at 15.9 and 16.4. Regardless of the technique used and of the inclusion or exclusion of samples from children with abnormal BPb, all of these estimates are, in the worst case, within 3 μg/dl of each other (range, 15.4–18.3). These findings clearly indicate that the threshold BPb for an increase of EP in urban children occurs between 15 and 18, with an average estimated value of 16.5. (A value of 16 may be the most valid because a range of 15.4–16.5 was obtained when the analysis was restricted only to those samples with "normal" BPbs.)

Probit analysis also estimates the dose at which 50% of the population responds. The natural occurrence of EP increases in children due to non-Pb-related causes (*C*) can be removed by using the Abbott formula (27) to compute the BPb at which 50% of the children show an effect exclusively from Pb exposure. By using this technique, the data were reevaluated. The BPbs at which 50% of the children had increased EP exclusively from Pb exposure were computed to be 29.9 and 35.2 for values that were >1 and >2 SDs above the mean of the reference group. (Corresponding values without the Abbott correction are similar—28.6 and 35.6, respectively.) An EP of >50 in a child with a BPb of >30 is presently considered an indicator of excessive exposure to Pb requiring further medical attention (14). Our study indicates that, at a BPb of 30, 27% of the children will have an EP of >53 (exceeding by 2 SD the mean of the reference group). Thus, even at the cutoff point of normal BPb, a substantial percentage of the population exhibits evidence of Pb-induced biochemical damage.

Increase of EP indicates an impairment of the completion of the synthesis of heme, a process that culminates with the insertion of iron at the center of the protoporphyrin molecule. Increased EP may result not only from Pb intoxication but also from a variety of other causes, such as chronic infections, certain hemolytic anemias, and FeD. The latter is the most frequent in children. FeD aggravates Pb intoxication both by increasing Pb absorption (28) and by enhancing the effects of Pb on heme synthesis. On the other hand, Pb intoxication leads to FeD by reducing iron absorption (29). These interreactions are, at least in part, mediated by the existence of a common carrier in the intestinal mucosa for both metals (30). In urban children, both FeD and Pb intoxication tend to occur with the greatest frequency in the same low socioeconomic class that is afflicted by both inferior nutrition and inferior housing. It can be hypothesized that, among urban children, those with FeD—and an inherent increase of EP—may have a tendency to absorb more Pb and, thus, to have BPb in the higher range of the "norm." In this case, the association of increased EP and BPb observed within the normal BPb range would be spurious, in reality reflecting only FeD. However, several arguments make this hypothesis implausible. First, EP is clearly correlated with BPb, with an exponential increase that continues at the same rate even when these reach extremely increased values. This relationship has been clearly documented not only in children (9–11), among whom FeD may occur frequently, but also in adult males (12, 13), among whom FeD is quite rare. Second, in the children studied by Roels and co-workers (26), the mean age was 10 yr (an age when FeD is rare), age and socioeconomic class of both groups were the same, and the only difference was in the proximity to a smelter, with the resulting increase in BPb. Third, because FeD is by far the most common cause of non-Pb-related increase of EP in children, its frequency is essentially identical to the value of *C* computed by probit analysis. At 2 SD, this was 2.4% in the present study (a value comparable to that observed in surveys of urban children of comparable age)—a frequency too low to influence the estimate of EP threshold in any significant manner. Fourth, in the present study, samples from 593 children age <2 yr were excluded to minimize the incidence of FeD, which is at its highest value in this age group. When the relationship between BPb and EP was analyzed, either in this group separately or by adding these samples to the rest of the data, the estimates of threshold remained essentially unchanged. Yet, the incidence of FeD in this group of children was substantially higher because the probit technique at 2 SDs yielded a *C* value of 4.5%. It appears, therefore, that the increase of EP is a direct effect of the increase in BPb. EP increases exponentially with a linear increase in BPb. A linear increase in BPb, in turn, reflects an exponential increase in body burden of Pb (7). Thus, the exponential increase in EP reflects an exponential increase in the dose of Pb.

Our study demonstrates a threshold BPb above which the frequency of an increase of EP is greater, which is well within the range of BPb currently called normal. Our observations, together with the reports of inhibition of AmLev dehydratase (22), confirm the hypothesis of Patterson that, even at the low level of exposure of the general population, adverse biological effects of Pb can be demonstrated if searched for with adequate techniques.

The clinical relevance of AmLev dehydratase inhibition is not limited to its effect on heme synthesis. The secondary accumulation of its substrate, AmLev, can affect the neurological system in a manner similar to the action of porphobilinogen (31, 32). The clinical symptomatology of Pb poisoning is close to that of acute intermittent porphyria, a disorder with accumulation of both AmLev and porphobilinogen (33). The accumulation of AmLev is reflected by its increased excretion in the urine, which is exponentially correlated with BPb (24). Selander and Cramer reported that urinary AmLev excretion accelerated at BPb >40, a value at the time accepted as the upper limit of normalcy (24). Because it is well established that AmLev dehydratase is significantly inhibited at BPb <40 (22), the failure to acknowledge any accumulation of AmLev at low doses of Pb made it necessary to postulate a "reserve capacity" of the enzyme (7). Yet it is apparent that the exponential increase in urinary AmLev starts in reality at BPb well below 40, once this *a priori* restriction is removed.

The effect of Pb on EP reflects its interference with the last step of the heme biosynthetic pathway, at the level of the mitochondria in the erythrocyte precursors in the bone marrow. Thus, the elevation of EP has the same biological meaning as increased urinary AmLev, that is, damage to a biochemical step beyond any hypothetical reserve capacity. The evidence of damage to a mitochondrial function is obvious in the erythrocyte because blood is the easiest tissue to sample. However, the affinity of Pb is not limited to the mitochondria in the bone marrow but also to those in other tissues (4). Inhibition of heme synthesis affects all body tissues in which heme is the prosthetic group of the cytochrome system. Accumulation of metalloporphyrins further inhibits mitochondrial function (34). The effect of Pb on protoporphyrin has been shown to occur in neural tissue cultures, where its increase is most obvious in the glial cells (35). Damage to glial cells is a common autopsy finding in childhood neuroencephalopathy (36).

The biochemical damage underlying certain clinical symptoms of Pb toxicity has been clarified recently [such as the inhibition of guanine aminohydrolase in saturnine gout (37) or the inhibition of adenylcyclase in neuroconductive disturbance (38)]. Biochemical damage to the heme synthetic pathway, which is reflected by inhibition of AmLev dehydratase and elevation of EP and has the potential of mediating neurological toxicity, cannot be discounted solely because it is already apparent at "normal" BPb, and it is not easily associated with clinically obvious symptoms. It is now clear that the BPb levels currently accepted as normal reflect instead a high level of environmental pollution (1–3). In fact, subtle signs of neuropsychological disturbance have been recently demonstrated at levels of exposure to Pb in the usual range in apparently normal suburban children by use of well-controlled experimental design (6).[¶]

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[¶] Yet the recent National Health and Nutrition Examination Survey (NHANES II) still demonstrates that, among American children age <5 yr, the frequency of individuals with BPbs at >30 is 4%. Among urban children, the frequency is 7.2%; in those residing in the central city, it is 11.6%. The average BPb (and the frequency of children with BPbs of >30) is inversely correlated to family income (Annest, J. L., O'Connell, D., Roberts, J. & Murphy, R. S., American Public Health Association, Los Angeles, CA, November 1–5, 1981, Abstr. 2125).

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EFFECTS OF AGE AND BODY LEAD BURDEN ON CNS FUNCTION IN YOUNG CHILDREN. II. EEG SPECTRA

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Low level lead (Pb) exposure has been shown to have behavioral effects in children (Otto et al. 1981), but there have been only unquantified case reports of EEG effects. Burrows et al. (1951), Bradley and Powell (1954), Byers and Maloof (1954), Thurston et al. (1955), Smith et al. (1963) and Fejerman et al. (1973) reported such symptoms as slowed alpha frequency, reduced EEG amplitudes, dysrhythmic activity and sometimes seizure-like activity. These reports involved high levels of Pb exposure which were associated with overt clinical symptoms. In one case where Pb values below 60 μg/dl were reported, EEGs were normal by visual classification (Smith et al. 1963).

EEG spectra in children were shown to be a function of age by Matousek and Petersen (1973). From 1 through 15 years, low frequency EEG amplitude diminished. Amplitude in alpha (α) spectral bands increased slightly over age. The peak frequency of rhythmic α waves increased as a function of age, first appearing at about 3 months in the 3.5 Hz range and approaching the 10 Hz range at 7–10 years.

Development of lateral dominance in EEG of children is less well documented due partly to methodological differences among investigators (Donchin et al. 1977). No consistent lateral dominance has been demonstrated in the EEG of children below the age of 11 years even though handedness appears at about 3 years (Giannitrapani et al. 1966; Joseph et al. 1976; Varner et al. 1977). Measures of cerebral dominance used in these studies were ratios or differences of amplitudes and phase relationships. These measures were computed using either total EEG amplitude, selected frequency bands or specific rhythmic activity such as the α wave.

Typically measures of laterality describe the relationship between *total* activity at each pair of sites, both synchronized and unsynchronized. Possibly the critical relationships are those between bilaterally synchronized waves only. It is possible, conceptually and analytically, to separate left and right hemisphere EEG into bilaterally synchronized waves and bilaterally unsynchronized waves (random with respect to each other). Least squares estimators have been defined for two sites of measurement which describe the relationship of the synchronized activity *only* and exclude all non-synchronized activity (Bendat and Piersol 1971). These measures include the 'coherence spectrum' which is one multiple correlation between synchronized portions of two EEG waves in each frequency band. The 'gain spectrum' is one regression coefficient for the relationship between the synchronized portions of two

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EEG waves in each frequency band. 'Phase' estimates the time delay between two synchronized waves in each frequency band.

Gain is interpretable as the amplification of a signal path between left and right hemisphere (Gersch and Tharp 1976) and coherence as a measure of signal-to-noise ratio (Bendat and Piersci 1971). Galbraith (1967), Busk and Galbraith (1975) and Livanov (1977) argued that when coherence is high between two CNS sites, then information is being transmitted between those sites. Busk and Galbraith (1975) showed that highest coherences were observed between sites known to have anatomical connections.

In children below 3 years of age, coherences have an average value of 0.2—0.3 with occasional peaks as high as 0.6 (Shulte and Bell 1973; Joseph et al. 1975; Livanov 1977). In adults, coherences of 0.4—0.7 are commonly reported (Livanov 1977; Shaw et al. 1979; Weller and Montagu 1979). Montagu (1975) reported essentially adult values (0.5—0.7) for children with mean age of 5.6 years. No data were found on normative values for interhemispheric gain spectra.

This study was designed to explore the relationships of age and PbB to children's EEG, employing spectrum analysis techniques. Results on power spectra were to be compared with age effects reported in the literature and to be evaluated for PbB effects. Spectrum analysis methods were used to provide sensitive new indices of laterality.

Methods

Subjects and data collection

Subjects were selected from the same group as used in Otto et al. (1981), except that only data from the frequent-rare (FR) paradigm were used. Because different data windows were used and fewer trials were required, more subjects were available in this analysis. A total of 37 males and 32 females (65 Blacks and 4 Whites) was used. Means, standard deviations and ranges of age, IQ (Bayley or Stanford Binet), socioeconomic scale (Hollingshead Two-Factor Index of Social Position), number of trials per subject and PbB levels are shown in Table I.

TABLE I

Demographic characteristics of sample.

	Mean	S.D.	Minimum	Maximum
Age (months)	36.8	17.5	13	75
IQ	85.9	14.0	46	118
Socioeconomic scale	66.9	13.4	22	91
Trials	14.2	5.3	4	24
PbB	29.5	13.3	6	59

Analog-to-digital conversion and editing were the same as in Otto et al. (1981) except that data from left and right parietal (P3 and P4 in the 10-20 system) were used for spectrum analysis. Subjects were included if 4 or more artifact-free trials were avialable. One second pieces of data were used, beginning 0.5 sec after each rare stimulus.

Data reduction

Data were analyzed by a variety of spectrum analyses using Hanning weights. A fast Fourier transform was computed from each 1.0 sec piece of EEG data from both P3 and P4. Spectrum measures, described below, where then derived from these raw spectra which were averaged over all available trials for each subject. The dependent variables were therefore one of each kind of spectrum per subject. Logarithmic transformed power spectra were computed from the raw spectra (Bendat and Piersol 1971) in order to meet normal theory requirements for hypothesis testing.

Cross-spectra and laterality

Regression analysis was used to obtain one set of least squares estimates of laterality for each subject. The slope and intercept using P4 log powers as the dependent variable and P3 log powers as the predictors were used as measures of relationship of P4 log power relative to P3. These terms are similar to averaged amplitude ratios and differences for each frequency band. Squared correlations between P3 and P4 log powers were used to describe the trialwise consistency of laterality for each subject.

Measures of relationship between bilaterally *synchronized* EEG (rather than total EEG) were also computed for each frequency band. The 3 spectra were the coherence, gain and phase between P3 and P4. These spectra were computed for each frequency band, from spectral quantities averaged across all trials (Benignus 1969).

Subjects in this study were awake with eyes open and since data were collected after a rare stimulus, very few high amplitude α waves developed. All of the measures of laterality as defined above, both for synchronized and total activity, are mathematically and physically meaningful even when no 'sinusoidal' wave activity is present.

Statistical analysis

Initially it was unclear how many frequency bands in the spectra should be used. To resolve this question all spectra were computed with the following 8 frequency bands: 1–2, 3–4, 5–6, 7–8, 9–10, 11–12, 13–16, and 17–20 Hz. A principal components analysis of a residual covariance matrix yielded an estimate of the structure of the dependent variables without the contaminating effects of the independent variables (Muller et al. 1979). After decisions about the principal components were made, statistical significance tests were made using the multiple regression technique described by Otto et al. (1981). Snooping procedures, such as principal component analysis, capitalize on chance relationships so that significance tests made during regression analysis are biased toward too many significant results. The high cost of a false negative and the cost and uniqueness of the data, made this approach necessary. Consequently, this study must be replicated before conclusions are firmly accepted. However, in view of the Bonferroni corrections applied to the repeated significance tests (see Table II), these results might still be viewed as more conservative than reported in other articles where uncorrected repeated tests are used.

A weighted least squares procedure was used to test a regression model with the spectrum measures as dependent variables. Observations were weighted by the square root of the number of trials for each subject in order to homogenize variances. Predictors were treated as 4 groups: (1) demographics (SES, IQ and sex), (2) age effects (linear, quadratic and cubic), (3) PbB effects (linear, quadratic and cubic), and (4) age by PbB interaction effects (age linear by PbB linear, age quadratic by PbB linear, PbB quadratic by age linear and age quadratic by PbB quadratic). Orthogonal polynomials for age and PbB were used to evaluate the linear, quadratic and cubic terms.

Each group of dependent variables was tested separately (e.g., gain for all bands). Each of the 4 groups of predictors was tested as a set in the order listed. If the first set was not significant, it was not included in the regression model, and the second set was tested. If a set was significant, stepdown tests were used to determine which variables in the set were to be included in the model. The ordering of sets was chosen so that demographic effects, if present, were controlled in tests of age and PbB, and effects of age were controlled in tests of PbB. Predicted trend lines and confidence intervals about the trend lines were produced for each model, as well as an estimate of the variance accounted for by the model.

Regression analysis was used to analyze these data because a continuous distribution of PbB values was observed. PbB values in this study ranged from 'normal' values to those slightly above normal (CDC 1978). Artificially dividing the sample into groups which could have been termed 'normal controls' and 'moderate PbB' would have been a less sensitive procedure. Regression analysis permitted continuous comparisons across the entire range of PbB values.

Results

Principal components

Principal components analysis showed that only 4 frequency bands were required to describe spectra. These bands were 1–4 Hz (δ), 5–8 Hz (θ), 9–12 Hz (α), and 13–20 Hz (β). Also, log power spectra from P3 and P4 could be averaged together to produce a single, more stable spectrum, P3 + P4. Two of the spectral measures of laterality, phase and intercept, accounted for little variation among subjects (principal components) and were thus eliminated from hypothesis tests.

Significance tests

The 5 spectra included in the significance tests were (1) P3 + P4 log power spectrum, (2) slope spectrum, (3) squared correlation spectrum, (4) gain spectrum, and (5) coherence spectrum. Table II shows results of stepwise multivariate tests for these variables. Each test is multivariate because 4 frequency bands were tested. Overall tests for the 5 groups of dependent variables showed a simple pattern in which no demographic variables and no interactions were significant. P3 + P4 log power spectra, gain and coherence were significantly affected by age. Gain was affected by PbB. Each of the results which was significant was further explored.

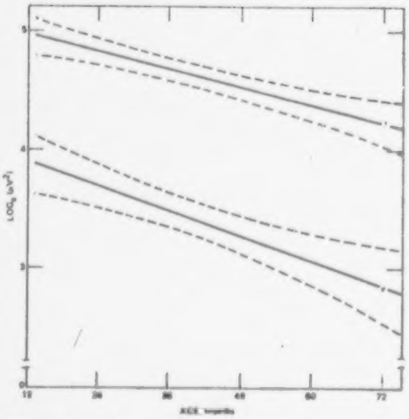

Fig. 1. P3 + P4 log power and 95% confidence bounds in the δ and θ frequency bands as a function of age. Estimated trend lines account for about 26% of variance in δ band, $P < 0.0001$, and about 22% in the θ band, $P < 0.0001$.

TABLE II

Results of multivariate tests from regression analysis. Numbers in the field of the table are P values. To keep family-wise type I error rate below $P < 0.1$ for each independent variable set, a P value in the table must be $P < 0.02$ in order to be declared significant (Kirk 1968).

	Demographic variables	Age	PbB	Pb by age interaction
P3 + P4 log power spectrum	0.31	0.0000003 *	0.26	0.15
Slope spectrum	0.69	0.12	0.052	0.12
Squared correlation spectrum	0.38	0.024	0.66	0.61
Gain spectrum	0.68	0.003 *	0.011 *	0.17
Coherence spectrum	0.12	0.0005 *	0.64	0.65

* Statistically significant.

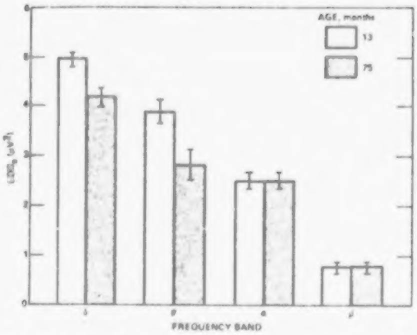

Fig. 2. Estimated P3 + P4 log power spectra and 95% confidence bounds for 13- and 75-month-old subjects.

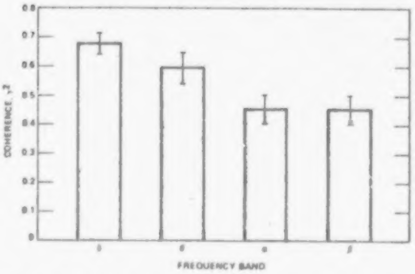

Fig. 3. Estimated grand mean coherence spectrum with 95% confidence bounds.

Power spectra and age

Stepdown tests of the effect of age on log power spectra showed only an overall significant linear effect in only the δ and θ bands. Fig. 1 shows that δ and θ band log power declines as a function of age from 13 to 75 months. The 95% confidence bounds about the predicted lines are also shown. The estimated relationship accounts for about 26% of the variance in the δ band and about 22% in the θ band (multiple $r \approx 0.5$). Fig. 2 shows estimated log power spectra for ages 13 and 75 months, the youngest and oldest ages in this study.

Coherence spectra and age

Results of planned subtests on coherence spectra, which were significantly affected by age in the overall test, showed that only linear effects were significant, but that no single coherence band was by itself significantly related to age. Inspection of the canonical variates revealed that δ and α band coherences decreased, while θ and β band coherences increased as a function of age. Trend lines in this finding, however, had wide confidence limits. Since this pattern is not readily interpretable nor very strong, the grand mean coherence spectrum was computed (Fig. 3).

Gain spectra, age and PbB

Gain spectra were affected by both age and PbB. Stepdown tests revealed only linear effects and that only the δ band gain increased as a function of age (Fig. 4). Fig. 5 shows a plot of the grand mean gain spectrum for the 3 higher frequency bands and a

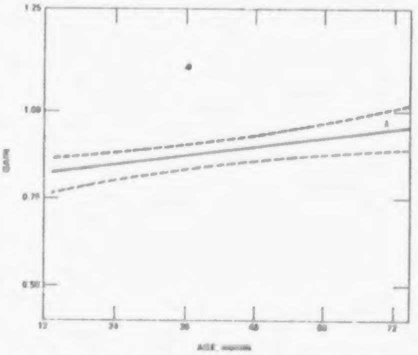

Fig. 4. Gain and 95% confidence bounds between P3 and P4 in the δ band as a function of age. Trend accounted for about 10% of the variance, $P < 0.01$. Values were estimated from the model for PbB values of 20 μg/dl.

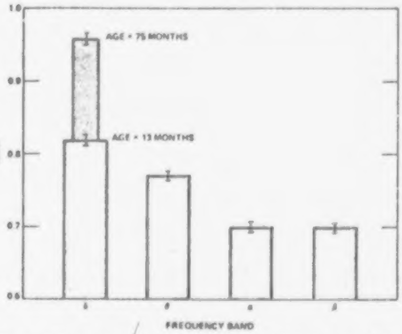

Fig. 5. Gain spectra with 95% confidence bounds for youngest and oldest ages in the study. Values were estimated from the model for PbB of 20 μg/dl. Only the δ-band coherence differed as a function of age.

double bar plot for the δ for 13- and 75-month-old subjects. These graphs were plotted for a PbB level of 20 μg/dl.

Evaluation of the effect of PbB on gain showed that only the cubic term was significant in all frequency bands. Fig. 6 shows a plot of the trend for gain in the δ band as a function of PbB, which is typical for all 4 bands. The gain had a sharp increase below approximately 15 μg/dl, leveled off, decreased slightly between about 30 and 40 μg/dl and then rose sharply after 40 μg/dl.

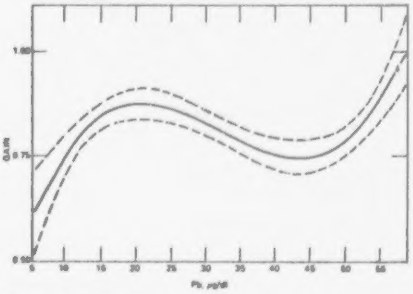

Fig. 6. Estimated cubic trend line and 95% confidence bounds for gain between P3 and P4 in the δ band as a function of Pb. Age was held at mean value of 36.8 months. Trend line accounted for about 20% of variance in δ band. Trends for θ, α and β bands were similar with variance accounted for of about 6%, 11% and 11%, respectively.

Since gain was below unity for all frequency bands and subjects, it was implied that P3 was always greater in amplitude of synchronized EEG than P4. Since the estimate of gain spectra is biased, however, it was necessary to test the gain spectra for P4 predicting P3, and compare the results to P3 predicting P4. This was done for several combinations of age and PbB. In all cases for all frequency bands, the gain as estimated for P4 predicting P3 was higher than for P3 predicting P4. This tended to confirm the conclusion that P3 amplitude is higher than P4 amplitude when only synchronized EEG is considered.

Discussion

Log power spectra

Comparison of Fig. 1 with data in Matousek and Petersen (1973) reveals a remarkable agreement. An exception is that they reported an early increase in α-band power which was not observed in this study. Their data were collected from bipolar P3-O3 and P4-O4 electrodes while the data here were collected from monopolar P3 and P4 to linked ears. They used nearly 15 times as many subjects as this study so that a smaller effect could have been significant. Also, since subjects in this study were measured in the eyes open, stimulated condition, little rhythmic α occurred (see Fig. 2). It was therefore impossible to verify the reported gradual increase in α frequency as a function of age. Note that despite the fact that children were used as subjects, adult frequency values were used to define the α band. Since no rhythmic α waves were observed this produced no problems.

Laterality

The classical measures of laterality, slope and squared correlation, were not significantly affected by age or PbB. The lack of age effects in these measures is in agreement with Giannitrapani et al. (1966), Joseph et al. (1976) and Varner et al. (1977).

It was shown that the δ-band gain increased linearly with age, but that the corresponding coherence did not vary with age in a similar or simple fashion. If it is assumed that coherence is constant over age, then perhaps signals are passed between hemispheres with more amplification as subjects mature. Another possibility is that some common signal source begins sending more signals to both hemispheres as age increases. Early δ activity, while of greater amplitude, is apparently more bilaterally random. As the subject matures the δ activity might be either brought under some common control or various sites might become more dependent upon each other. Whether this should be viewed as an increase in interhemispheric processing, a decrease in lateral specialization under these stimulation conditions, or some other change in function is not clear without further data.

It is noteworthy that for all subjects, regardless of age or PbB, the amplitude at P3 was estimated to be greater than at P4 for *synchronized* EEG. This represents an EEG laterality even in children well below 75 months in age. This tends to support the notion that the relative *synchronized* EEG amplitude is functionally important since behavioral handedness is reported to be present in children of this age range. Other measures of EEG laterality have not shown hemispheric differences in young children.

The magnitude of gain in all frequency bands was affected by PbB. While the effects followed a cubic trend and decreased as a function of PbB for mid-range values, the tendency was for gain to increase as a function of PbB of extreme values. In view of the number of subjects and the stepwise data analysis, it would not be wise to take the exact cubic trend too seriously without replication. It is, however, apparent that low or high PbB is associated with increases in gain in all frequency bands. Possibly as PbB levels approached 15 μg/dl, some bodily compensatory mechanism was brought into action which for mid-range PbB values managed to offset effects. As higher PbB values occurred, the hypothetical compensatory mechanism was overcome and the PbB effect increased. As noted above, functional interpretation of gain spectra is, at present, not clear.

The conclusion that gain is affected by PbB levels below 15 μg/dl is potentially important since it is one of the lowest-level PbB effects reported in the literature. If the functional interpretation of interhemispheric gain can be discovered, then PbB effects on behavior might be better understood. Since the effects are apparently present at 'normal' PbB levels, it is important that: (1) the functional nature of interhemispheric gain be explored, and (2) the PbB effect be replicated.

It is likely that PbB values observed in this study are lower than peak values which occurred in the past in these subjects. If that is so, then the relatively low PbB level effects shown in this study are in fact due to a higher Pb exposure in the past. The epidemiologic nature of this study precludes an answer to that question. The direction and nature of EEG effects of Pb in children have been documented, however, even if the exact PbB history is unknown. That none of these children had shown clinical signs of Pb poisoning implies that even at their maximum PbB levels their poisoning was not very severe.

Summary

This study explored the effects of age and PbB upon EEG power spectra and various measures of hemispheric laterality in children, aged 13—75 months, watching a display (cartoon). The following are the principle conclusions:

(1) The δ- and θ-band amplitude decreased with age.

(2) When only bilaterally *synchronized* EEG between P3 and P4 was considered, the amplitude of P3 was estimated as greater than P4 in all frequency bands and for all ages. Previous reports have not shown lateral EEG dominance in children below 75 months.

(3) When lateral dominance measures consider only the relationship between *synchronized* EEG at P3 and P4, bilateral communality in the δ band increased with age.

(4) Increased PbB generally produced an increase in the relative amplitude of *synchronized* EEG between P3 and P4 in all frequency bands. This was true for PbB levels well below 15 µg/dl, among the lowest level PbB effects previously reported. No clinical or behavioral effects of PbB values have been reported below 15 µg/dl.

It appears to be theoretically and practically important to understand the functional significance of bilaterally synchronized activity. The signal processing of the CNS can be explored using these methods. Greater understanding of these data would help define the extent and etiology of PbB effects on the CNS.

Résumé

Effets de l'âge et de la surcharge corporelle en plomb sur le fonctionnement du système nerveux central chez des jeunes enfants. II. Spectres EEG

Cette étude explore les effets de l'âge et de la surcharge en plomb (SPb) sur les spectres de puissance EEG et diverses mesures de latéralité hémisphérique chez l'enfant âgé 13—75 mois, en train de regarder une séquence visuelle (dessin animé). Les données suivantes sont les conclusions principales:

(1) L'amplitude de la bande delta et thêta diminue avec l'âge.

(2) Quand on considère seulement l'EEG *synchronisé* bilatéral entre P3 et P4, l'amplitude en P3 apparaît supérieure à celle en P4 dans toutes les bandes de fréquence et pour tous les âges. Les rapports antérieurs n'ont pas montré de dominance latérale EEG chez des enfants au-dessous de 75 mois.

(3) Quand les mesures de dominance latérale considèrent seulement la relation entre les EEG *synchronisés* en P3 et P4, la communauté bilatérale dans la bande delta augmente avec l'âge.

(4) La SPb produit généralement une augmentation de l'amplitude relative de l'EEG synchronisé entre P3 et P4 dans toutes les bandes de fréquence. Ceci est vrai pour des niveaux de SPb très inférieurs à 15 µg/dl, qui constituent les niveaux les plus bas jamais rapportés auparavant. Aucun effet clinique ni comportemental des valeurs du SPb n'ont été rapportés au-dessous de 15 µg/dl.

Il semble être important, d'un point de vue théorique et pratique, de comprendre la signification fonctionnelle de l'activité synchronisée bilatérale. Le traitement du signal du système nerveux central peut être exploré en utilisant ces méthodes. Une meilleure compréhension de ces données aiderait à définir l'étendue et l'étiologie des atteintes du système nerveux central par le SPb.

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Neuroepidemiologic Evaluations of Children with Chronic Increased Lead Absorption

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A fortuitous union of the techniques of neurotoxicology and epidemiology has recently led to a greatly increased understanding of the neurotoxic properties of inorganic lead. Lead has been shown to cause a dose-related spectrum of neurologic impairment, which ranges in severity from encephalopathy to subtle derangement of intellect, behavior, and peripheral motor function (25). The range of this impairment has been found to extend downward to a level of exposure which only a decade ago was considered safe (1,7,9,27,30,31). There remain differences of opinion as to the possible existence of thresholds for the neurotoxicity of lead and as to the shape of dose-response relations (22). However, debate today concerns not whether lead levels in children of 80 versus 60 μg/dl are without danger, but whether neuropsychologic dysfunction begins to supervene at blood lead levels of 50 or 30 μg/dl or at even lower concentrations (36).

Epidemiologic analysis of the acute and chronic neuropsychologic changes caused in children by lead has, like many such analyses (10,11), proceeded through three fairly distinct stages. The first stage was that of clinical recognition—in this case the recognition by Byers and Lord (6) that acute lead exposure in children could produce chronic neuropsychologic dysfunction. That clinical observation received initial corroboration through a series of case-control and cross-sectional evaluations (9,27) in which neurotoxicologic and epidemiologic techniques were combined. More definitive corroboration was then provided by highly sophisticated, blind cohort analysis, such as that of Needleman and his colleagues (24), which demonstrated most conclusively the chronic neurotoxicity of lead in children and indicates that this toxicity may become evident at levels of exposure below those heretofore considered harmful.

In 1973 and 1974, our group at the Center for Disease Control (CDC) conducted two cross-sectional epidemiologic investigations into the neuropsychologic consequences of chronic increased exposure to lead (18,20). Both studies were undertaken in groups of children living near lead smelting plants. These studies have been corroborated and to an extent superseded by later work. They illustrate, however, an historical stage in the development of current concepts concerning low level lead toxicity. In addition, they document the serious, but thoroughly correctable hazard that is posed to children by the metallic emissions of lead smelting plants.

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EL PASO STUDIES

Epidemiologic Background

The first of our studies was conducted in El Paso, Texas, near a large ore smelter that has been in operation since 1887 and which has the capacity to extract lead, copper, and zinc from over 800,000 metric tons of ore concentrates annually (19). In December, 1971, the El Paso City–County Health Department found that this smelter had emitted 1,012 metric tons of lead, as well as 508 tons of zinc, 11 tons of cadmium, and 1 ton of arsenic into the atmosphere through its stacks in the years 1969 through 1971.

Environmental sampling in El Paso in 1971 and 1972 indicated that there was extensive contamination of air, dust, and soil with lead and other heavy metals. The highest levels for each element were found at the smelter, and lead levels above background were found in surface soil for as far as 10 km from the smelting plant. The mean annual airborne lead concentration immediately downwind of the smelter in 1971 was 92 $\mu g/m^3$.

To determine the extent of human lead absorption, we conducted a random-sample survey of blood lead levels in August, 1972, among families in the 13 census tracts closest to the smelter. We divided the survey area along census tract lines into three roughly concentric circles with the smelter at the center and with outer radial distances of 1.6, 4.2, and 6.1 km. We obtained interviews in 670 (80.4%) of the 833 selected occupied households.

In participating families, we obtained venous blood samples from all persons 1 to 19 years of age. Blood lead levels were determined at CDC by modified Delves' cup atomic absorption spectrophotometry (4). Full-thickness samples of interior paint and samples of household dust and of surface yard soil were obtained for lead analysis at each home. Any pottery vessel used for food storage was tested for lead release (16).

The highest blood lead levels were found in area 1, and within that area, the highest levels were noted in Smeltertown, the section of the city most closely adjacent to the smelter (Fig. 1). Levels elsewhere in area 1 were intermediate between those in Smeltertown and those in areas 2 and 3. These differences were statistically significant ($p < 0.001$).

The most severe lead uptake was found in children 1 to 4 years old. In

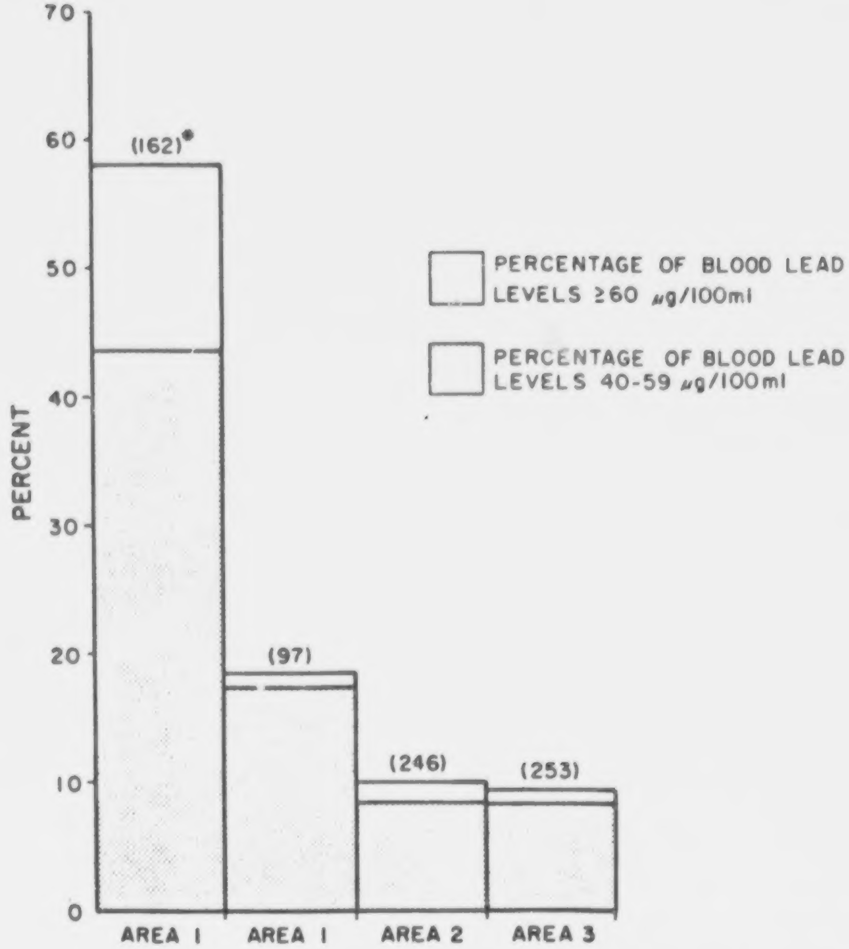

FIG. 1. Distribution of blood lead levels in persons 1–9 years old, by area; El Paso, Texas, August, 1972. Numbers within parentheses indicate number of children tested. Area 1 on the left represents Smeltertown only; area 1 on the right does not include Smeltertown.

area 1, 55% of the children in that age bracket had lead levels of 40 to 59 µg/dl.[1] Another 14% had levels ≥ 60 µg/dl. In areas 2 and 3, 27% of the 1–4-year-old children had levels ≥ 40 µg/dl, with 4% ≥ 60 µg/dl. We did not carry out systematic testing beyond area 3. However, complementary data (26) from Ciudad Juárez, Mexico, immediately across the international boundary from El Paso, indicate that blood lead levels above background were to be found in children living as far as 12 km from the El Paso smelter. Elevated blood lead levels in El Paso were not restricted to children 1 to 4 years of age. Forty-five percent of the 5–9-year olds and 31% of the 10–19-year olds in area 1 also had blood levels ≥ 40 μg/dl.

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[1] A whole-blood lead level of 40 µg/dl or more was previously considered by the Surgeon General (32) to be indicative of "undue lead absorption," and a confirmed level of 80 µg/dl or more to represent "unequivocal lead poisoning." On the basis of more recent data on the subclinical toxicity of lead, the "undue absorption level" had been revised downward to 30 µg/dl (33).

Exposure to lead-based paint was approximately equal in each of the three study areas in El Paso and did not correspond to the distribution of lead in blood. Exposure to lead-containing pottery was equally uniform and occurred at only two homes in area 1, seven in area 2, and three in area 3. Such exposures may occasionally have produced increased lead uptake but cannot have accounted for the overall geographic pattern of lead absorption.

Concentrations of lead in dust, soil, and air, on the other hand, closely paralleled the distribution of lead in blood. In area 1, there was a strong quantitative relationship between blood lead levels and exposure to lead in house dust ($p < 0.001$) and in soil ($p < 0.05$). These data indicate that ingestion by children of lead deposited in dust and soil by the smelter had been a major pathway of lead uptake in El Paso (29). Airborne lead also appears to have been readily available for absorption, and airborne lead levels in Smeltertown were certainly sufficient to have produced substantial transpulmonary uptake (35).

Neuropsychologic Evaluations

To determine whether children exposed to lead near the El Paso smelter had suffered subclinical impairment of central or peripheral nervous system function, we conducted neuropsychologic evaluations in 1973 (20).

All children included in the neuropsychologic study were 3 years 9 months to 15 years 11 months old and had lived within 6.6 km of the El Paso smelter for at least 12 of the 24 preceding months. All except three had participated in the epidemiologic survey; the three exceptions were sibs of epidemiologic survey participants. Excluded from analysis were any children with a history of symptoms compatible with acute lead poisoning or of lead encephalopathy and children who had at any time received ethylenediaminetetraacetate (EDTA) chelation therapy. Included were four children who had received oral penicillamine therapy, but whose blood-lead levels had not fallen below 40 μg/dl on repeated determinations.

Participating children were divided into two groups. In the lead-absorption group were all those who in 1972 had a blood-lead level of 40 to 80 μg/dl, as well as three children not tested in 1972 whose blood-lead levels in 1973 exceeded 40 μg/dl. The control group consisted of children whose blood lead levels had been < 40 μg/dl in August, 1972. Controls were selected so that as a group they matched children in the lead absorption group for age, sex, language spoken at home (English or Spanish), length of residence within 6.6 km of the smelter, census tract of residence in August, 1972, and socioeconomic status (15).

After parental permission had been granted, children in the two groups were evaluated individually by examiners who did not know the children's blood lead levels or group assignments. Children first underwent complete medical and neurologic evaluations. No cases of symptomatic lead poisoning were found, but five children—two in the lead-absorption group and three controls—were found to have preexisting, presumably unrelated neurologic disease and were not studied further. No gross impairment was found on neurologic examination of any of the other children in either group.

The parents of those remaining were asked whether their children had pica or had experienced recurrent abdominal pain, clumsiness, irritability, or convulsions. Parents of children 10 years or younger were asked to rate their child's level of activity according to a standard scale (39). All children's behavior and activity during the examination were also rated by the physician.

Neurologic Assessment

All children 5 years old and above were given the following neurologic tests intended to evaluate motor function:

1. Standing on each foot (recorded as the greatest number of seconds up to 15 in the best of three trials).
2. Tandem walking (recorded as the most correct steps up to nine in the best of three attempts).
3. Alternate tapping with a stylus on two metal plates 30 cm apart, a measure of proximal arm muscle function and of coordination (recorded for each hand as the number of taps in one 10-sec trial after a 5-sec warm-up period).
4. Tapping with a stylus on a single metal plate while hand and wrist were held above the table (finger-wrist tapping), a measure of wrist flexor and extensor muscle function (recorded for each hand as the number of taps in one 10-sec trial after a 5-sec warm-up period).
5. Visual reaction time (recorded for each hand in msec as the average of three trials).
6. Auditory reaction time (recorded for each hand in msec as the average of three trials).

Electronic testing equipment similar to that developed by Baloh (3) was used in tests (3–6).

Psychologic Assessment

Psychologic evaluations were done by one of six psychometrists. The Wechsler intelligence scale for children (WISC) (37) was used for 34 children, 5 years 1 month old and above, in the lead-absorption group, and for 36 children above age 5 years in the control group. The Wechsler preschool and primary scale of intelligence (WPPSI) (38) was used for 12 preschool children, 3 years 9 months to 5 years 7 months, in the lead group, and 15 preschool children, 3 years 9 months to 6 years 6 months, in the control group.

All psychometrists were bilingual, and tests were administered in English or Spanish, as appropriate for each child; 15 (33%) children in the lead-absorption group and 19 (24%) children in the control group were tested in Spanish ($p > 0.5$ by chi square). The following subtests were used in the WISC: information, arithmetic, comprehension, and digit span in the verbal scale, and picture completion, block design, object assembly, and coding in the performance scale. Subtests used in the WPPSI were information, arithmetic, comprehension, block design, animal house, and geometric design in the performance scale. The vocabulary subtest was omitted from the verbal scale of both WISC and WPPSI because of the poor standardization that results on translation of this subtest into Spanish. All Wechsler scores were age standardized (37,38). As a further measure of perceptual motor function, all children were administered the Bender motor gestalt test (5), which measures accuracy in copying simple geometric designs; accuracy was scored using an age-standardized scale (17). The behavior of each child in the psychologic testing situation was evaluated by the psychometrist using a standardized rating scale (23).

A venous blood sample for lead analysis was obtained from every child at the time of neuropsychologic testing. Analysis was performed in duplicate by modified Delves' cup atomic absorption spectrophotometry (4) and results were expressed as the mean of the two determinations.

TABLE 1. *Demographic features; El Paso, Texas—1973*

Characteristics	Lead absorption group	Control group
Number	45	76
Blood-lead (μg/dl):		
mean (and range), 1972	48.3(40–68)	26.9(1–39)
mean (and range), 1973	40.5(22–58)	26.5(15–39)
Age (yr)[a]	8.3 ± 3.4	9.3 ± 3.6
Age distribution		
<5	10(22%)	12(15%)
5–8	17(37%)	25(32%)
9–11	10(22%)	18(23%)
12–15	9(20%)	23(29%)
Male/female	30/16(65%/35%)	45/33(58%/42%)
Number speaking some or all Spanish at home	45(98%)	74(95%)
Socioeconomic index[a]	66.9 ± 13.7	66.4 ± 15.0
Duration of residence in study area (yr)[a]	6.6 ± 3.4	6.7 ± 3.6
Site of residence, 1972		
0–1.6 km from smelter	22(48%)	35(45%)
1.7–4.0 km from smelter	16(35%)	29(37%)
4.1–6.6 km from smelter	8(17%)	14(18%)

[a] Mean ± SD.

TABLE 2. *Medical history data; El Paso, Texas—1973*

	Prevalence rates	
Category	Lead absorption group	Control group
Pica	7(15%)	4(5%)
Abdominal colic	12(26%)	11(14%)
Clumsiness	5(11%)	6(8%)
Irritability	18(39%)	21(27%)
Convulsions	3(7%)	3(4%)
Hyperactivity score[a]	0.88 ± 0.79	0.69 ± 0.70

All results nonsignificant by *t*-test.

[a] Mean ± SD.

Mean age in the lead-absorption group was 8.3 (± 3.4) years and in the control group 9.3 (± 3.6) years (Table 1). This difference was not statistically significant ($p > 0.10$). Sixty-five percent of the lead group and 58% of the controls were male. The groups matched closely in ethnic background, primary language, socioeconomic index (15), and duration of residence near the smelter.

Pica, colic, clumsiness, irritability, and convulsions were all reported by parents to have been more frequent among children in the lead absorption group than among controls (Table 2). None of the observed differences were, however, statistically significant. Neither hyperactivity nor other behavioral abnormality was significantly more common in either group as measured by a parental questionnaire (39) or by the physician's evaluation.

Finger-wrist tapping was significantly slowed in the dominant hand of children

TABLE 3. *Results of quantitative neurologic tests; El Paso, Texas—1973*

Test	Lead absorption group	Control group
Visual reaction time[a]		
Dominant	29.2 ± 9.9	26.3 ± 8.8
Nondominant	29.2 ± 8.6	26.3 ± 10.2
Auditory reaction time[a]		
Dominant	25.5 ± 9.2	24.7 ± 9.4
Nondominant	26.8 ± 9.0	24.0 ± 7.2
2-Plate tapping[b] (no. of taps)		
Dominant	15.9 ± 8.3	15.9 ± 3.9
Nondominant	14.7 ± 7.5	14.9 ± 4.1
Finger-wrist tapping[b]		
Dominant	46.4 ± 13.4[c]	54.1 ± 12.3
Nondominant	41.2 ± 12.4	45.5 ± 10.8

[a] Expressed in msec.

[b] Number of taps are given.

[c] $0.001 < p < 0.01$ by one-tailed *t*-test.

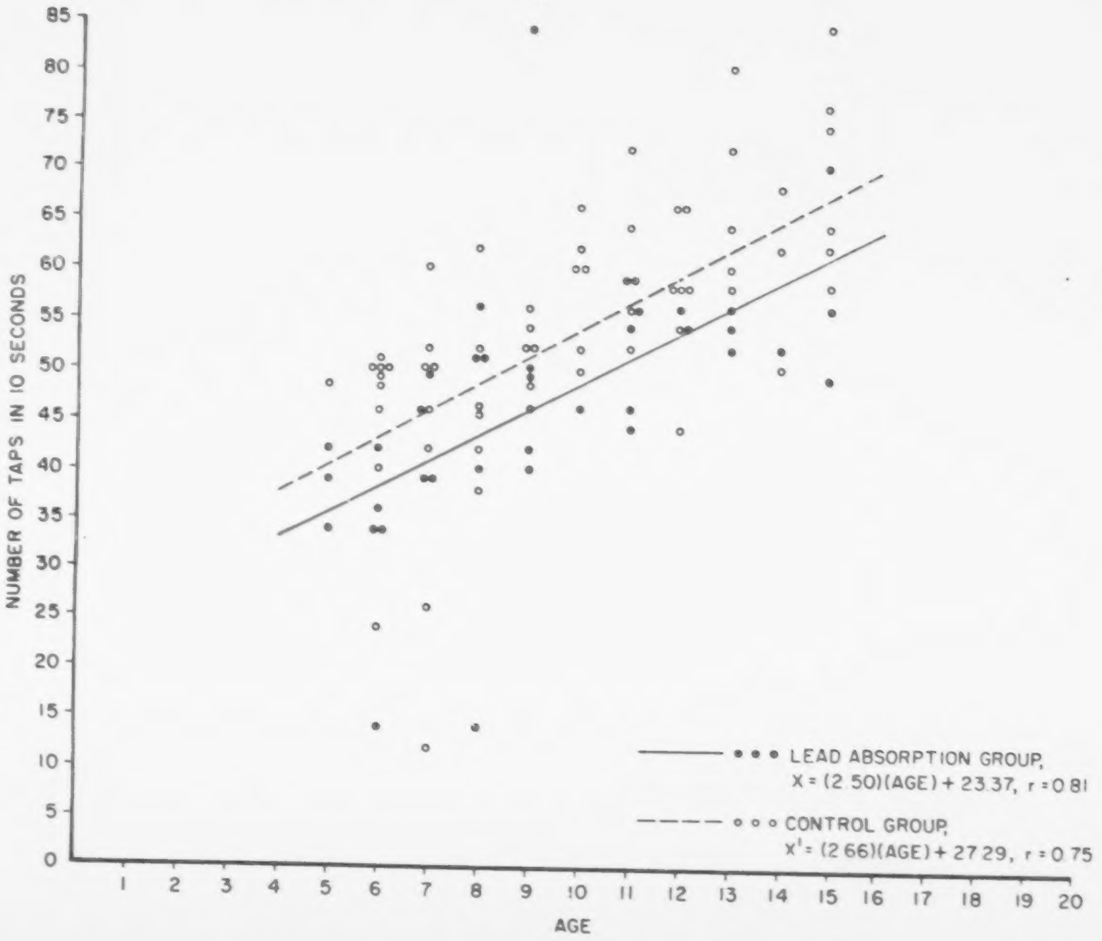

FIG. 2. Regression of finger-wrist tapping speed (dominant hand) by age and study group; El Paso, Texas, June, 1973.

in the lead absorption group (46.4 taps in 10 sec versus 54.1, $p < 0.01$) (Table 3). To adjust those data for age, a regression of dominant hand finger-wrist tap data was plotted against age for each group (Fig. 2); the slopes of the resulting lines are nearly parallel, indicating that the difference between groups persisted at all ages. No other significant differences were found between the groups in quantitative neurologic testing (Table 3).

Psychologic Data

Full-scale IQ scores and verbal IQ scores showed no significant differences between the groups (Table 4). Results in the Bender gestalt test (5) and in the psychologists' evaluation of behavior (23) also showed no significant differences ($p > 0.05$). Analysis of performance IQ scores showed, however, that children in the lead absorption group scored significantly below children in the control group in both the WISC and the WPPSI taken separately as well as in the two tests combined (Table 4). Mean performance IQ scores with WISC and WPPSI combined were 94.9 for the lead absorption group and 102.7 for the control group ($p < 0.01$). This difference resulted from the accumulation of differences in each of the eight subtests administered (Fig. 3); the most highly significant differences were found in the WISC coding subtest and in the WPPSI geometric design subtest. The difference between the groups in performance IQ persisted when the groups were partitioned by sex and when children with a history of pica were excluded from analysis.

TABLE 4. *Mean Wechsler intelligence test scores; El Paso, Texas—1973*

Test	Lead absorption group	Control group
WISC		
Verbal IQ	83.47 ± 11.71	85.30 ± 15.27
Performance IQ	96.44 ± 13.74[a]	103.29 ± 17.87[a]
Full scale IQ	88.68 ± 12.64	93.29 ± 16.11
WPPSI		
Verbal IQ	84.92 ± 11.60	84.47 ± 12.39
Performance IQ	90.67 ± 11.64[a]	100.27 ± 11.29[a]
Full scale IQ	86.17 ± 11.17	91.20 ± 11.88
WISC ± WPPSI		
Verbal IQ	83.85 ± 11.57	85.14 ± 14.69
Performance IQ	94.93 ± 13.35[b]	102.71 ± 16.79[b]
Full scale IQ	88.02 ± 12.21	92.88 ± 15.34

[a] $0.01 < p < 0.05$ by one-tailed *t*-test.
[b] $0.001 < p < 0.01$.

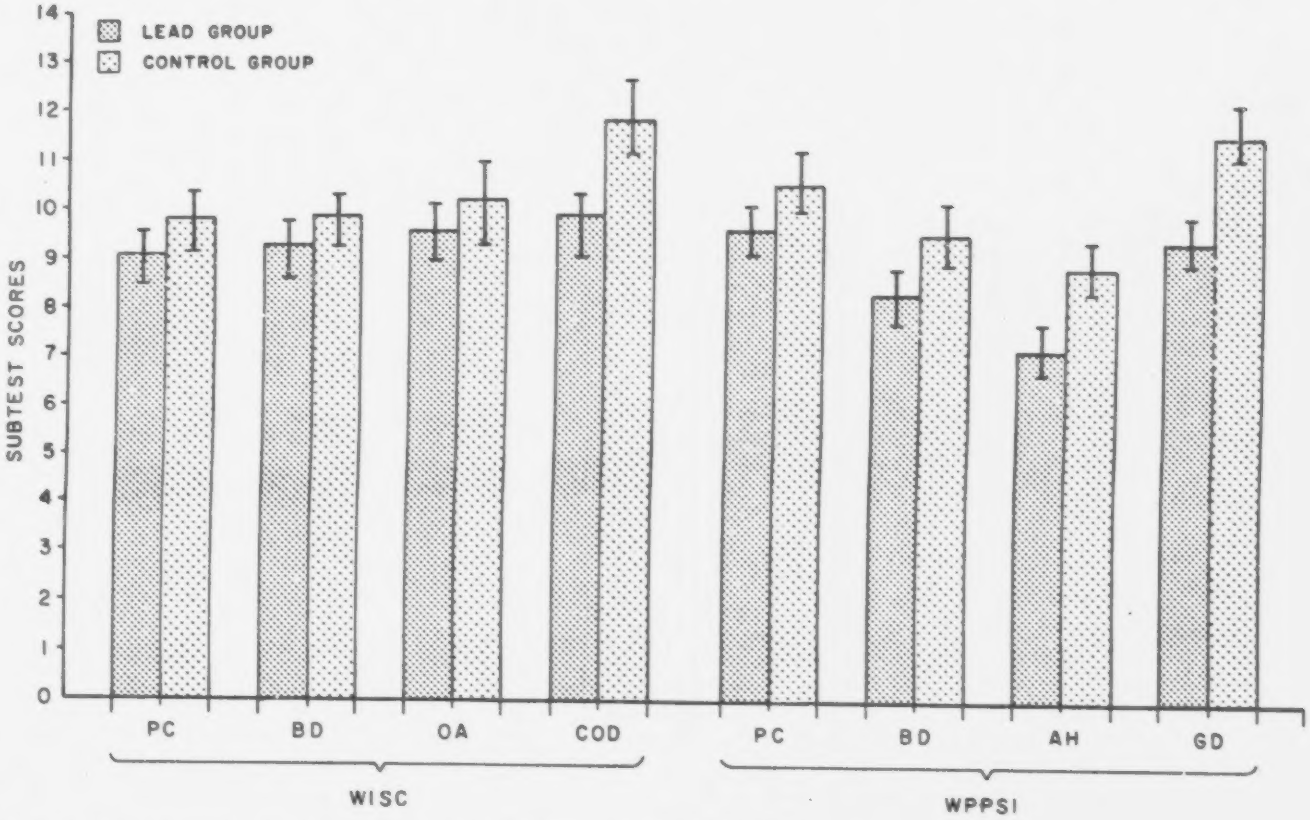

FIG. 3. Wechsler performance scale subtest scores in lead-exposed and control children; El Paso, Texas, 1973. *(Bars)* ± 1 SE. Subtests: picture completion (PC), block design (BD), object assembly (OA), coding (COD), animal house (AH), and geometric design (GD).

IDAHO STUDIES

Epidemiologic Background

The second of our studies was conducted in 1974 among children who lived near an ore smelter in Kellogg, Idaho (18). This smelter is located in a narrow, intermountain valley in the northern Idaho panhandle. Average emissions of lead are estimated to have been approximately 10 metric tons per month from 1955 through September, 1973, when they increased abruptly to 35.3 tons per month following a fire in the main filtration unit.

Environmental sampling near this smelter indicated that air lead levels had risen from 1971 to 1974 in parallel with smelter emissions. Highest environmental lead levels were found immediately at the plant; in July, 1974, the mean air lead level at the smelter boundary was 18 $\mu g/m^3$. Peak levels of lead in surface soil and vegetation in 1974 were 9,000 ppm and 3,418 ppm, respectively.

We undertook our epidemiologic studies in August, 1974, after two children (siblings, 2 and 3 years old) were hospitalized with chronic unexplained abdominal pain, maximal blood lead levels of 68 and 89 $\mu g/dl$ and "lead lines" in their long bones. We employed the same concentric study design that we had used in El Paso (19), but the geographic scope was broader. There were five concentric strata, with outer radial distances of 1.6, 4.0, 10.0, 24.0, and 32.0 km. In addition, we included in the survey a geologically similar side valley, which branched from the main valley 20 km (12 mi) from the smelter and which was intended to be a geologic control, that is, an area with natural lead in the soil but with no airborne contamination. In fact, however, visible pollution was noted there each morning during the survey and thus the area's value as a control was limited. Finally, we examined a rural control area 72 km (45 mi) northwest of the smelter. Complete interviews were obtained in 588 (86%) of the 686 occupied houses that had been selected for survey. Procedures for collection and analysis of samples were similar to those used in El Paso (19).

We found that 385 (41.9%) of the 919 1–9-year-old children examined in the five study areas had blood levels of 40 to 79 $\mu g/dl$, and an additional 41 (4.5%) had blood lead levels ≥ 80 $\mu g/dl$ (Fig. 4). In the area closest to the smelter, all but 2 (98.8%) of 172 children evaluated had lead levels ≥ 40 $\mu g/dl$, the highest being 164 $\mu g/dl$. The prevalence rate for blood lead levels ≥ 40 $\mu g/dl$ decreased slowly with distance from the smelter (Fig. 4). In the geologic control area, the prevalence of levels ≥ 40 $\mu g/dl$ was 10.2%. In the rural control area, 1 (1.1%) of 89 children examined had a blood level ≥ 40 $\mu g/dl$. No clinical cases of lead poisoning were noted. In all areas blood lead levels were higher in the 1–4-year age group than in the 5 to 9.

The relationships between lead in blood and levels of lead in air, soil, and dust at the Idaho smelter were similar to those noted in El Paso, except that there was a somewhat closer correlation between blood lead and air lead levels ($r = 0.59$). No significant correlation was evident between blood lead levels

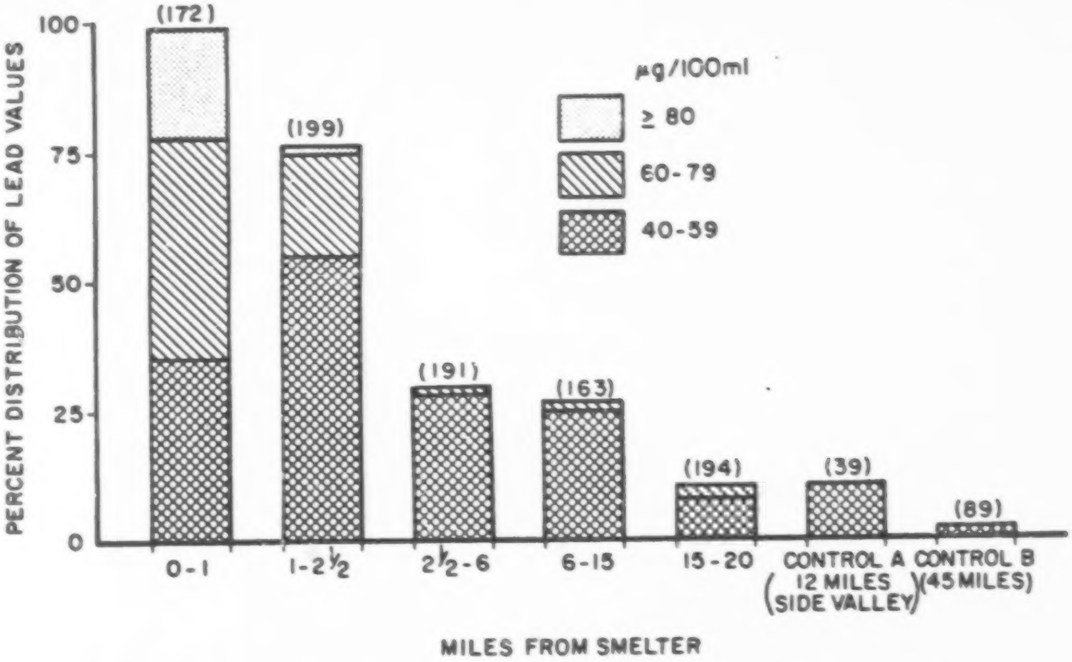

FIG. 4. Distribution of blood lead levels in 1–9-year-old children near north Idaho smelter, August, 1974. Lead values are given in μg/100 ml. Numbers within parentheses indicate number of children tested.

and paint lead exposure, and pottery was used for food storage in only 5 (0.9%) of the 588 homes visited.

Neurologic Studies

To evaluate the possible occurrence of subclinical neurologic changes in children living near the Idaho smelter, we conducted studies of neurologic function in October, 1974, 2 months after the conclusion of the epidemiologic studies.

We evaluated 183 5–9-year-old children from the six study areas (including the "geologic control" area) and 39 children from the rural control area. All children first received blind, standardized medical and neurologic examinations. Seventeen children (7 of them with blood lead levels ≥ 40 μg/dl) were found to have preexisting medical or neurologic lesions and were not studied further. Also excluded were 3 children on whom we obtained incomplete data. The remaining 202 were all apparently healthy, and none had clinical evidence of neurologic abnormality.

Peripheral nerve conduction velocity was measured in the right peroneal nerve of each of these children at a standardized room temperature of 21 to 24°C using cutaneous electrodes and without sedation (12). At least three supramaximal stimuli were delivered at proximal and distal locations along the nerve trunk, and at each location the most reproducible (modal) latency between stimulus artifact and muscle action potential was recorded. No frankly pathologic conduction velocities were noted. However, when conduction velocity in each child was compared with blood lead level, a statistically significant negative correlation was found between the two ($r = -0.38$) (Fig. 5). A similar negative correlation was found between conduction velocity and erythrocyte protoporphyrin (EP) concentration (14,28) ($r = -0.40$). Correlations between conduction velocity and age, sex, and socioeconomic status were not significant.

DISCUSSION

We concluded from these studies that the ore smelters in El Paso, Texas, and in Kellogg, Idaho, were major localized sources of environmental contamination, and that these plants had been responsible for widespread increased absorption of lead by nearby children.

The data indicate further that chronic increased absorption of particulate lead from the smelters, sufficient to produce blood lead levels of ≥ 40 μg/dl, had caused subtle but statistically significant impairment in neurologic and in psychologic function in exposed children. In the El Paso studies we observed diffuse impairment in the nonverbal cognitive and perceptual motor skills (13) measured by the performance scale of the Wechsler intelligence tests (37,38). Also, we detected impairment in the fine motor skills measured by the finger-wrist tapping test. In Idaho, although none of the children studied had clinical

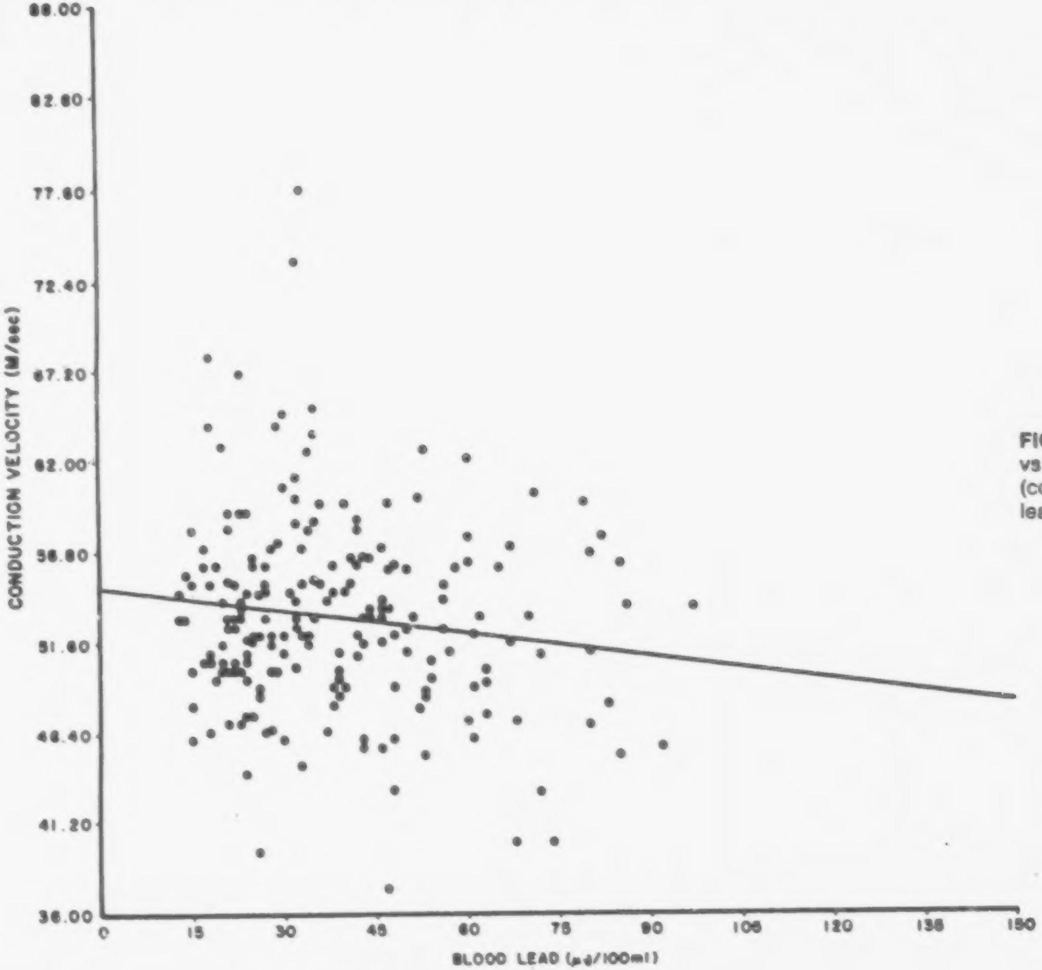

FIG. 5. Peroneal nerve conduction velocity vs blood lead level, Idaho, 1974. *y* (conduction velocity) = 54.8 − 0.45 × (blood lead) (*r* = −0.38) (*n* = 202).

neurologic disease or frankly pathologic conduction velocities, we observed in them a statistically significant dose–effect relation between decrease in maximal motor nerve conduction velocity and increase in blood lead level.

These results need careful interpretation, since the observed differences could have been produced by variation between the groups in matching characteristics, particularly in age or in sex (Table 1). In defense of the data, it should, however, be noted that performance IQ scores in the El Paso studies were age-adjusted by standard techniques (37,38), and that the difference in finger-wrist tapping persisted when scores were stratified by age (Fig. 2). The performance IQ differences also persisted when the groups were stratified by sex. There is the possibility that the observed impairment could have preceded and perhaps predisposed to the lead absorption. Pica, which may be a manifestation of subtle neuropsychologic impairment, was more common in the lead group in El Paso than in the controls. However, the differences between the high and the low lead groups in performance IQ and in finger-wrist tapping persisted when those children without a history of pica were examined separately.

The results of these studies are consistent with much of the large body of data on chronic, low level lead toxicity that has been generated over the past decade by case control and cross-sectional evaluations both of workers exposed occupationally to lead (1,2,7,25,30,31) and of exposed children (10,27). These studies have now received strong corroboration through the cohort analyses of Needleman and his colleagues (24). Despite the occasional appearance in the literature of a negative study, the data on low level lead toxicity have been sufficiently convincing to deliberative and regulatory bodies in the United States and abroad (34,35,40) that these agencies have in recent years promulgated increasingly stringent recommendations concerning reduction of lead exposures (33). In their review of this topic (35), the U.S. Environmental Protection Agency concluded that "surprisingly low levels of blood lead can at times be associated with the most extreme effects of lead poisoning, including severe, irreversible brain damage," and further that "evidence tends to confirm that some type of neural damage does exist in asymptomatic children and not necessarily only at very high levels of blood lead." The clear implication to be drawn from these data is that lead exposure, particularly of children, ought to be reduced to the lowest limit possible. Lead is ubiquitous in our society (22), and exposure to lead from many sources, even from such a high dose source as lead-based paint (8), will be controlled only with great difficulty. There are, however, sources of lead that are readily controllable at the source. Chief among these are lead emissions from automobiles and emissions from the lead industries. It has been demonstrated in El Paso since 1972 that lead emissions from the smelter there can be controlled and that through such primary prevention the blood lead levels of nearby children can be reduced (21). Such a demonstration provides heartening documentation of the economic and technical feasibility of reducing children's exposure to lead through attainment of air lead standards (35).

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Erythrocyte Nucleotides in Children—Increased Blood Lead and Cytidine Triphosphate

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Summary

The erythrocyte nucleotides of 25 children, 1–5 years old, with normal and increased blood leads, were assayed by anion-exchange high-performance liquid chromatography. Red blood cells of the 12 children with blood lead (PbB) below 30 μg/dl (20.3 ± 6 μg/dl, $\bar{X}$ ± S.D.) had normal levels of activity of pyrimidine 5′-nucleotidase (P5N) and their erythrocytes were virtually free of pyrimidine nucleotides except for small amounts of UMP and UDP. The purine nucleotides, predominantly ATP and GTP, were present at values similar to adults. In the 13 children with PbB 30–72 μg/dl (45.3 ± 14.3 μg/dl), total cytidine phosphates (CMP, CDP, CTP) were significantly ($P < 0.001$) increased from trace values to 8.31 ± 6.21 nmoles/10^{10} erythrocytes. The purine nucleotides were unchanged. P5N activity was 143.3 ± 22.0 units/g hemoglobin in children with PbB < 30 μg/dl and 75.4 ± 24.2 units ($P < 0.001$) in the high lead subjects. There was a logarithmic correlation of erythrocyte cytidine phosphates with PbB ($r = 0.89$, $P < 0.001$) and an inverse correlation of cytidine phosphates with *ln* P5N activity ($r = 0.59$, $P < 0.001$), of *ln* P5N with PbB ($r = 0.64$, $P < 0.001$) and of *ln* P5N with *ln* erythrocyte zinc protoporphyrin (Protoporphyrin IX) ($r = 0.57$, $P < 0.001$).

Speculation

The accumulation of small amounts of erythrocyte CTP in children with increased lead exposure but with blood concentrations of leads as low as 30 μg/dl supports a lower threshold for the consequences of lead induced inhibition of pyrimidine 5′-nucleotidase (P5N) than indicated by earlier, less sensitive assays of nucleotides in adults with lead poisoning. The presence of CTP as the predominant pyrimidine nucleotide is similar to the profile in congenital deficiency of P5N but the nucleotides accumulate with less suppression of P5N than found in congenital deficiency. The significance of increased red cell cytidine phosphates at low levels of lead exposure is unknown but it appears to relate to suppression of P5N activity early in red cell maturation, and thus provides an index of antecedant lead exposure that correlates with erythrocyte zinc protoporphyrin.

It is generally accepted that the human erythrocyte is virtually free of pyrimidine nucleotides and is characterized by a selective, possibly unique, enzyme system that favors conservation of the purine nucleotides which serve as the precursors of ATP (25). It was Valentine *et al.*'s (23) description of a congenital hemolytic anemia characterized by the intraerythrocyte accumulation of pyrimidine nucleotides that led to the definition of a pyrimidine 5′-nucleotidase (P5N) (E.C. 3.1.3.5) in the erythrocyte cytosol. P5N specifically dephosphorylates the pyrimidine nucleoside monophosphates OMP, UMP, and CMP, allowing diffusion from the cell as the soluble nucleosides: cytidine and uridine (14).

Congenital deficiency of P5N, typically with less than 10% of normal enzyme activity, is relatively frequent among the definable causes of nonspherocytic, familial hemolytic anemia (11, 12, 23). It may be manifested by early and recurrent jaundice, has a possibly increased association with mental retardation (6) and is characterized by a basophilic stippling of the erythrocytes resembling lead poisoning. Of analogous interest, P5N is readily inhibited by lead both *in vitro* and *in vivo* (14). Clinical studies by ourselves (2, 3, 9, 11) and others (8, 11, 16) have established the logarithmic suppression of erythrocyte P5N evident at even low levels of blood lead, but the effect of this partial inhibition of P5N on the erythrocyte nucleotides of children has not previously been reported. This is also the first report, employing high performance liquid chromatography technology, of the normal nucleotide profile of young children.

In this study the erythrocyte nucleotides of 25 children, 1–5 years old, were assayed by anion-exchange high performance liquid chromatography to define the values in control subjects and in those previously identified by the Omaha Lead Paint Poison Prevention Program as having an excessive lead burden. At the time of the testing, 13 children had blood leads in the range of 30–72 μg/dl but none had anemia or reticulocytosis. Assays in these 25 children and 22 others were utilized to define the intercorrelations of blood lead, cytidine phosphates, P5N, and erythrocyte protoporphyrin IX (zinc protoporphyrin or ZPP).

MATERIALS AND METHODS

The participants in the study were 47 children 1–5 years old followed in the Omaha Lead Paint Poison Prevention Program because of a previous blood lead (PbB) over 30 μg/dl or erythrocyte zinc protoporphyrin (ZPP) over 60 μg/dl and 22 healthy children of the same age range seen in our outpatient clinic. Not all blood samples were adequate for all assays and the distribution of the number of subjects for each of the various combinations of assays was as follows:

Assay	Lead Project	Out-Patient	Total
ZPP, P5N	25	22	47
PbB, P5N	20	22	42
PbB, Nucleotides	19	6	25
PbB, P5N, Nucleotides	7	5	12

Because of the significant correlations of data with the standard indices of lead absorption, PbB and ZPP, the analyses according to the two classes of subjects are not incorporated in the data presentation. Informed parental consent was received for all studies. The study was annually approved by the peer professional review committee of the University of Nebraska Medical Center.

Before nucleotide assay the red cells were first freed of platelets and white cells by elution with 5 volumes of saline from 15 ml syringes packed with equal parts of microcrystalline cellulose and alpha cellulose at 4°C (5, 19). All assays were performed on venous blood.

Nucleotides were extracted from the erythrocytes by the method of Chen, *et al.* (9), which involves erythrocyte hemolysis in distilled water, protein precipitation with 10% trichloracetic acid, and neutralization of the trichloracetic acid with tri-N-octylamine in Freon 113 (Matheson Gas Products, Inc.) after centrifugation. The aqueous extracts were immediately frozen at −17°C. Samples were analysed within 5 days after extraction but found to be stable at this temperature for several wk. The extraction efficiency was calculated to be 85–100% for all nucleotides.

The chromatographic system for nucleotide analysis consisted of two model 110 A pumps and a model 420 microprocessor from Altex Scientific, Inc., Berkeley, CA; a model 1203 fixed wavelength UV detector operating at 280 nm from Laboratory Data Control, River Beach, FL; and a variable volume sample injector from Rheodyne, Inc., Berkeley, CA. Chromatographic columns were 25 × 0.46 cm Partisil 10 SAX anion exchange columns from Altex Scientific, Inc., Berkeley, CA.

Nucleotide separation was accomplished by gradient elution with a low concentration eluent of 0.007 M KH_2PO_4 at pH 3.90 and a high concentration eluent of 0.25 M KH_2PO_4 and 0.50 M KCl at pH 4.50. An isocratic period of 6 min followed by a series of short linear gradient steps forming a 35 min concave gradient from 0–100% enabled complete 5′-nucleotide separation in less than 70 min. The column was then re-equilibrated with 45 ml of the low concentration eluent. The flow rate was constant at 1.5 ml/min and the column was at ambient temperature.

Chromatographic peaks were identified by comparison with reference standards from Sigma Chemical Co., St. Louis, MO. Well resolved separations were obtained of the following 5′-nucleotides: the monophosphate ribonucleosides of cytidine (CMP), uridine (UMP), adenosine (AMP), guanosine (GMP), inosine (IMP) and xanthosine (XMP); the di- and triphosphate ribonucleosides of cytidine (CDP and CTP), uridine (UDP and UTP), adenosine (ADP and ATP) guanosine (GDP and GTP), and inosine (IDP and ITP). Quantitation of the nucleotides was accomplished by relative peak areas of the known standards to the internal standard XMP.

P5N activity of erythrocytes was assayed by the method of Torrance, *et al.* (21). One unit is defined as the enzyme activity that will hydrolyze 1 nmole ^{14}CMP/min/g hemoglobin. Whole blood lead was measured by atomic absorption spectroscopy by the microcup method (10). Although red cell lead is a better index of the lead burden than whole blood lead, particularly in anemic subjects, the decision was made to utilize whole blood lead as the more universal standard of exposure. Erythrocyte zinc protoporphyrin was measured by the method of Lamola (13) employing an ESA Zn P Model 4000 hemofluorometer (Cambridge, MA). One ZPP unit is defined as the presence of 1 g of zinc erythrocyte protoporphyrin/dl whole blood. All values are given as the mean ± S.D.. Differences in mean values for normal data were analyzed for statistical significance by a one-tailed Student's *t* test with the Mann-Whitney U test used for non-normal data.

RESULTS

The chromatographic separation of the erythrocyte nucleotides identified the 13 major 5′-nucleotides.

Blood samples from the six outpatients, considered to be control subjects had either no cytidine phosphates present (four children) or less than 0.25 nmoles/10^{10} red blood cells (rbc) (two subjects). As shown in Figure 1, the presence of measurable amounts of cytidine phosphates increased with the blood lead and cytidine phosphates were found in all 10 samples from children with blood leads of 37–72 μg/dl. Cytidine phosphates were found in 11 of 13 samples from children with a blood lead ≥30 μg/dl but in only four of 12 samples with a blood lead <30 μg/dl.

Since a blood lead of 30 μg/dl is the current level considered indicative of excessive lead exposure, the nucleotide profiles were examined for these two groups. (Table 1). The 13 children with a PbB ≥30 μg/dl (30–72 μg/dl) had a mean PbB of 45.3 ± 14.3, significantly higher than the 20.3 ± 6.0 μg/dl of the 12 children with a PbB of 8–29.9 μg/dl. The content of the purine nucleotides, of IMP and UDP and UMP were comparable for the two groups and resembled the nucleotide profile of normal adults. The ATP content of 1322.2 ± 279.6 nmoles 10^{10} rbc was similar to the 1204.7 ± 323.1 of the low lead subjects and to the generally accepted adult value of 1000 nmoles/10^{10}. The only difference in the erythrocyte nucleotide profiles between the two groups was the significant ($P < 0.001$, Mann-Whitney U test) increase in the cytidine phosphates, primarily as CTP, in the samples from the 13 children with PbB ≥30 μg/dl, in contrast to the trace values of the 12 children with PbB <30 μg/dl. In the high lead group, CTP was increased to 10.64 ± 10.65 nmoles/10^{10} rbc.

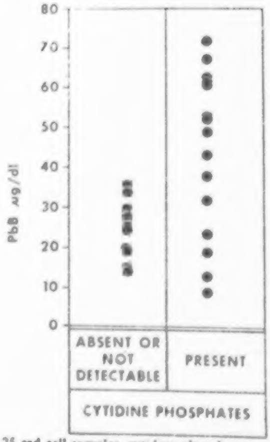

Fig. 1. Of 25 red cell samples, cytidine phosphates were found in 15. All subjects with blood leads >38 μg/dl had measurable amounts. No cytidine phosphates were found in the erythrocytes of eight of 12 children 1–5 yr old with a blood lead (PbB) <30 μg/dl; measurable amounts were found in 11 of 13 samples from children with blood lead ≥30 μg/dl.

Table 1. *Erythrocyte nucleotides nmoles/10^{10} RBC, $\bar{X}$ ± S.D. Children 1–5 yrs old*

Nucleotide	PbB ≥30 μg/dl $n = 13$	PbB <30 μg/dl $n = 12$
ATP	1285.17 ± 320.40	1209.17 ± 341.27
ADP	216.0 ± 160.02	152.73 ± 49.45
AMP	56.67 ± 17.68	30.3 ± 11.13
GTP	109.15 ± 49.72	81.96 ± 35.65
GDP	31.04 ± 31.72	21.14 ± 3.59
GMP	2.92 ± 1.45	15.4 ± 27.60
IMP	64.5 ± 54.34	65.96 ± 38.49
CTP	10.64 ± 10.65[1]	0.02 ± 0.07
CDP	3.88 ± 5.87	0.00 ± 0.06
CMP	0.56 ± 0.84	0.29 ± 0.50
UTP	1.27 ± 2.39	0.60 ± 0.00
UDP	2.44 ± 2.75	0.27 ± 0.85
UMP	5.87 ± 8.67	6.73 ± 9.96
PbB μg/dl	45.3 ± 14.3[1]	20.3 ± 6.0

[1] $P < 0.001$, Mann-Whitney U test.

Samples from 15 children, 11 of whom had PbB ≥30 μg/dl, had measurable amounts of cytidine phosphate. As shown in Figure 2, there was a significant logarithmic correlation of total erythrocyte cytidine phosphate content, in nmoles/10^{10} rbc (Y) with PbB μg/dl (X): $\ln Y = 2.44 \ln X - 7.13$; $r = 0.89$, $P < 0.001$. This regression predicts the accumulation of 329 nmoles of cytidine phosphates at PbB 200 μg/dl.

The correlation of red cell P5N (Y) and blood lead (X) as determined in 42 children (Fig. 3) again established a significant inverse logarithmic correlation: $\ln Y = 5.062 - 0.016X$; ($r = 0.64$; $n = 42$; $P < 0.001$). In 27 children with a PbB <30 μg/dl, P5N activity was 143.1 ± 22.0 units/g hemoglobin (mean ± S.D.), which is similar to our laboratory normal of 148 ± 20. In 15 children with PbB ≥30 μg/dl. P5N (mean ± S.D.) was 75.4 ± 24.3 or 53% of the low lead group.

As shown in Figure 4, P5N was also inversely correlated with ZPP, the index of lead exposure most commonly employed in community programs. Eight of the nine children with ZPP >60 μg/dl had significant decreases in P5N below the normal range of 128–168 units. The regression equation of the correlation of P5N (Y) and ZPP (X), $\ln Y = 5.3939 - 0.23 \ln X$ ($n = 47$; $r = 0.57$; $P < 0.001$) predicts a decrease of P5N to 45 units as ZPP increases to 1000 μg/dl.

The correlation of the lead-induced suppression of P5N (X) with accumulation of cytidine phosphates (Y) was investigated in the data from 12 children with measurable amounts of cytidine phosphates: $Y = 59.14 - 11.48 \ln X$; $r = 0.80$; $P < 0.001$ (Fig. 5). This regression equation predicts an increase in total erythrocyte cytidine phosphates to only 41 nmoles/10^{10} when P5N is reduced to 5 units or to less than 5% of normal.

DISCUSSION

The red cell nucleotides of children are shown in this study to be quantitatively and qualitatively similar to those of adults. Red cells are normally free of pyrimidine nucleotides except for small amounts of UDP. In this study of children 1–5 years old, detectable increases in pyrimidine nucleotides, primarily as cytidine triphosphate, correlated with increases in the blood lead and concomitant suppression of the pyrimidine-specific 5′-nucleotidase of the red cell cytosol (P5N).

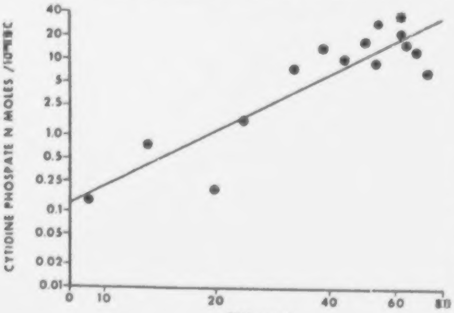

Fig. 2. Total red cell cytidine phosphates in nmoles/10^{10} rbc (Y) as related to blood lead (PbB, X axis) in the 15 children, 1–5 yr old, with measurable amounts of cytidine nucleotides. $\ln Y = 2.44 \ln X - 7.13$; $r = 0.89$; $P < 0.001$. This regression predicts 329 nmoles of cytidine phosphate at a PbB of 200 μg/dl.

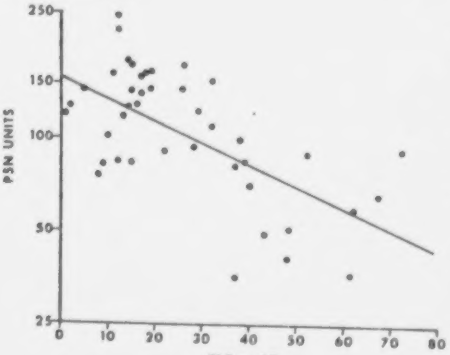

Fig. 3. Correlation of erythrocyte pyrimidine 5′-nucleotidase (P5N; Y) in units defined as the hydrolysis of 1 nmole CMP/min/g hemoglobin with blood lead μg/dl (PbB; X) in 42 children 1–5 yr old: $\ln Y = 5.062 - 0.016 X$; $r = 0.64$; $P < 0.001$. This regression predicts P5N activity of 1.8 units at a PbB of 200 μg/dl.

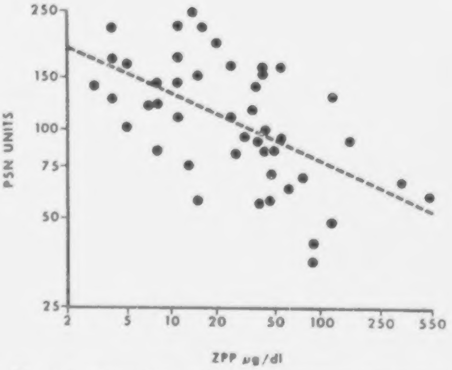

Fig. 4. Correlation of P5N activity (Y) with erythrocyte zinc protoporphyrin μg/dl (ZPP, X) as predicted by data from 47 children. $\ln Y = 5.3929 - 0.23 \ln X$; $r = 0.57$; $P < 0.001$.

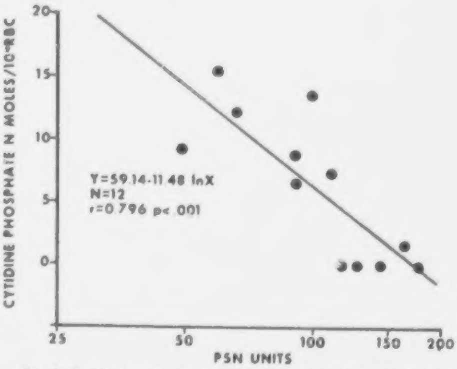

Fig. 5. Correlation of total red cell cytidine phosphates nmoles/10^{10} rbc (Y) with erythrocyte P5N (X) in 12 children, 1–5 years old: $Y = 59.14$ $11.48 \ln X$; this predicts 41 nmoles cytidine phosphate when P5N activity is reduced to 5 units.

The sensitivity of the high performance liquid chromatography assay of red cell nucleotides made it possible to show the accumulation of cytidine phosphates at blood leads as low as 30 μg/dl and at P5N activity of approximately 30–70% of normal. In congenital deficiency of P5N, spectrophotometric assays have not shown pyrimidine nucleotide accumulation until enzyme activity has been suppressed to below 10%; in adults with severe, symptomatic lead poisoning, pyrimidines were found when P5N was reduced to 15–16% activity (16). The slope of the observed correlation of PbB and P5N (Fig. 2) predicts negligible, or 10%, activity of P5N at a blood lead of 147 μg/dl or a red cell lead of 210 μg/dl. This is comparable to the prediction by Paglia *et al.* (16) of complete suppression of enzyme activity at 200 μg/dl of red cell lead. Inasmuch as our previous high performance liquid chromatographic assays of red cell nucleotides in lead-poisoned rabbits showed quantitatively similar responses to this childhood study, apparent differences in the sensitivity of adults and children for accumulation of pyrimidine nucleotides by lead-induced suppression of P5N are thought to relate to the method of assay. The effect of lead-induced suppression, however, may be both quantitatively and qualitatively distinct from the sequelae of congenital deficiency of P5N.

The observed correlation of PbB and cytidine phosphates (Fig. 1) predicts an increase in cytidine phosphates to only 329 nmoles/10^{10} rbc at a PbB of 200 μg/dl despite a concurrent prediction of almost complete suppression of enzyme activity. This is far lower than the massive increases of pyrimidine nucleotides to over 1500 nmoles/10^{10} rbc in persons found homozygous for deficits of P5N activity (6, 12, 23, 25). This would suggest qualitative differences in the sequelae of the two modes of decreased P5N activity as well as differences that may relate to a sustained suppression of P5N throughout erythrocytic maturation in congenital deficiency *versus* a relatively transient suppression induced by the daily variabilities in blood lead.

The distribution of the erythrocyte pyrimidine nucleotides in children with lead poisoning shows a greater increase in cytidine than uridine phosphates and a predominance of CTP. This is similar to the nucleotide profile noted by Torrance and Whittaker (22) in a child with congenital deficiency of P5N and by our laboratory (19) in studies of lead-treated rabbits. The presence of CTP as the dominant pyrimidine nucleotide is thought to reflect the relative rate of product formation of P5N for OMP > UMP > CMP (15). Continued activity of the diphosphokinases would favor the conversion of CMP → CDP → CTP but any postulated preference for this over the conversion of UMP → UDP → UTP requires delineation of the enzyme kinetics in the red cell (1).

Although Boninsegna *et al.* (7) suggest that the soluble fraction of the erythrocyte is unique among blood cells in the activation of UDP → UTP by a zinc sensitive enzyme that is distinct from adenylate kinase, there are no comparable studies of erythrocyte activation of CDP → CTP. Similarly, additional evidence is required in the mature red cell of the amination of UTP to CTP by CTP synthetase.

In the absence of evidence for a significant *de novo* synthesis of CTP in the mature erythrocyte, Valentine's view of the pyrimidines as residual to RNA of the normoblast is a more tenable hypothesis concerning the origin of the erythrocyte pyrimidine nucleotides. This is also consistent with our previous studies in lead poisoned rabbits (19), in which lead poisoning induced rapid *in vivo* suppression of P5N with a lag of almost 30 days before cytidine phosphates were detected in the red cell. The accumulation of pyrimidine nucleotides and of stippling peaked at 60 days, the time required in the rabbit for a complete turn over of circulating erythrocytes.

The concept of residual RNA as the source of the pyrimidines is also compatible with the basophilic stippling that is characteristic of both lead-induced suppression and congenital deficiency of P5N. Basophilic stippling is generally accepted as an aggregation of ribosomal RNA around the red cell mitochondria (4, 17, 18). The precipitation of unstable ribosomes requires the availability of divalent cations or oxidant activity to effect nucleic acid cross-linkage. Beutler (6) implies that lead poisoning and P5N deficiency may have the common denominator of an excess of divalent cation since the red cell stippling of P5N in both states is reversed by EDTA. It can also be proposed that an excess of pyrimidine nucleotides in the young red cell inhibits ribosomal degradation as well as effecting an abnormal charge that would favor aggregation. The simultaneous inhibition of ribosomal degradation by lead may favor ribosomal instability and precipitation at lesser levels of pyrimidine nucleotide accumulation than found in congenital deficiency.

The presence in red cells of a cytosolic nucleotidase (P5N) that specifically dephosphorylates the pyrimidines, facilitating their diffusion with simultaneous conservation of the purines ATP and GTP, would suggest the possibility that even low levels of pyrimidine nucleotides may exert a toxic effect. It is agreed that lead-induced nucleotidase deficiency can be sufficiently severe to mimic genetic impairment (25). Although the significance of the accumulation of red cell cytidine phosphates at low levels of lead exposure is unknown, it does appear to require suppression of P5N early in red cell maturation and thus provides an index of cumulative lead exposure during the genesis of the circulating red cells. The mechanism is distinct, but the time lapse is similar to the increase in erythrocyte zinc protoporphyrins due to inhibition of heme synthesis.

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The Identification and Elimination of a Potential Lead Hazard in an Urban Park

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ABSTRACT. During development of a park in Oakland, California, a potential public health hazard due to lead in the soil (approximately 1,000 to 100,000 ppm) was identified through cooperative sampling and analysis efforts of the staffs at the county and state health departments. Analysis of samples collected at various locations and depths in the park, coupled with a three-dimensional model of the lead values obtained, indicated steps necessary to eliminate the hazard. The county undertook the abatement program, and with the elimination of the health hazard, the park development has been completed. Concomitant blood lead screening of neighborhood children failed to establish the park as a source of elevated lead in any of the children tested.

EXCESS BODY BURDENS of lead (Pb) in humans are associated with a variety of health problems which can have severe individual and societal consequences.[1] There is mounting evidence that subtle neurological damage which can cause hyperactivity and learning and behavioral disabilities, occurs at blood Pb levels much lower than those which cause obvious clinical manifestations of poisoning.[2-7] The United States Center for Disease Control (CDC) recommends clinical management for children with blood Pb levels $\geqslant 30\ \mu g/dl$.[8] This management includes reduction of exposure, general pediatric care, family education, chelation therapy when appropriate, and correction of any existing nutritional deficiencies.

Children, particularly those with pica are most susceptible to an increased body burden of Pb. One important environmental source of Pb is contaminated soil. It is very difficult to determine the minimum level of contamination which presents a health hazard because of the many confounding variables, i. e., accessibility of soil to children, solubility of the particular Pb species in the soil, variability of children's behavior patterns, nutritional status and other Pb sources which contribute to the child's blood Pb level (water, paint chips, diet, inhalation of vehicle emissions, etc.). Duggan and Williams[9] assessed the risks from Pb content of street dust and estimated the highest tolerable concentration to be 300 ppm. The Lead Data Analysis Task Force of the Ontario Ministry of the Environment, in its assessment of problems near Pb processing plants in Toronto, concluded that "children who live in an area where the soil lead level is greater than 1,000 ppm have statistically significantly higher mean blood lead levels than do children who live in areas where the soil lead level is less than 600 ppm."[10] Further the Task Force recommended "cleaning of soil, street dirt, and house dirt be carried out in contaminated areas contiguous to the lead plants. These contaminated areas [are] to be designated as the zones in which the Pb content of the top two inches of soil exceeds 1,000 parts per million." For comparison, the U.S. Consumer Product Safety Commission bans products containing paints with more than 600 ppm Pb.[11]

Methods and Materials

Background

A wet-cell battery manufacturing plant on several acres of land located in a below-average income residential neighborhood of Oakland, California, was operational from 1924 to 1974. In 1976 the facilities were demolished and removed, including approximately the first 0.5 m of soil beneath the plant's concrete floor. A project to develop the land into a park to be used by children and adults was well underway when, in 1978, it was halted due to the concern of local residents and city and county officials over possible soil Pb contamination. Approximately fifteen samples were collected for analysis by private laboratories. Lead values for these samples were scattered over a large range with a maximum of 1,500 ppm. The County Health Care Services Agency then requested the State Department of Health Services to assist them in further investigations.

Sampling Strategy

Two questions required resolution: (1) was there a potential public health hazard? and (2) if so, what was the best way to eliminate it?

The first question was resolved in the following manner. A preliminary set of fourteen soil samples was collected at various locations on the property, with particular emphasis on locations where operational parts of the plant had been located. Approximately half the samples were taken from the surface and half from a depth of approximately 15 cm (since there were no records to establish clearly that all the contaminated soil had been removed when the plant had been demolished). Contaminated soil beneath the surface could pose a problem if overturned during the final landscaping process or during use of the park. The intial survey yielded a range of values from 10 to 10,700 ppm, the highest from a sample collected at 15 cm depth. These results coupled with previous knowledge of the existence of the battery plant made it clear that a comprehensive survey should be made.

Figure 1 is a schematic diagram of the park. Approximately one-half the park area, namely, that next to 96th Avenue, had been completed and consisted primarily of a recreational center, an asphalt playground, and a sand box. Lead values from these areas were all low. This was consistent with the fact that the battery plant had been located on the other half of the park adjacent to 98th Avenue.

A 24-point grid was constructed over the 98th Avenue half of the park in the area of concern, the distance between grid points being 6 m (Fig. 1). Samples were collected at the surface and at 15 and 30 cm depths. A total of seventy-one samples were collected and analyzed (one sample was lost during analysis). Horizontal contour maps for the surface, 15 and 30 cm, were constructed for the entire park using the values from these seventy-one samples plus the previous fourteen. Since the type of hazard reduction program depends on such parameters as the vertical distribution of Pb, and whether the contamination is localized, a three-dimensional representation of the horizontal and vertical contours of the Pb distribution of the grid area was constructed. This coupled with cost and landscaping considerations was then utilized to determine the appropriate hazard reduction procedure.

Sampling, Analytical, and Computational Methods

Most soil samples were collected using a 46-cm-long steel, chromium-plated sampling tube with an outside and inside diameter of 2.8 and 2.5 cm, respectively. The tube was cleaned after each sampling to avoid cross contamination of the samples. The extracted corings were separated by depth in the field and placed in lead-free plastic bags. Soil samples were air dried, rocks and vegetation were removed, and the remaining soil was powdered by shaking in a Spex mill for 5 min. Samples of 4 g were extracted with 20 ml of 10% HNO_3 (1.5 N) for 1 hr at 100°C. Extracts were analyzed by atomic absorption spectrometry using the 2833 Å resonance line described in AIHL Method 51.[12]

An estimate of the variability introduced due to nonuniformity in the milled samples was made by reweighing, extracting, and analyzing unused portions of ten randomly chosen samples. The second analysis gave 75% ± 10% of the Pb found in the previous analysis.

To estimate the effectiveness of our Pb extraction procedure, nine soil samples from the park were anlyzed separately by the Air and Industrial Hygiene Laboratory (AIHL), and the Department of Soils and Plant Nutrition at U.C. Berkeley. The samples were air dried, rocks and vegetation removed, and the soil ground in a mortar to pass a 2 mm screen. In the U.C. procedure, 5-g samples were extracted with 30 ml of 4 N HNO_3 for 16 to 24 hr at 70°C while AIHL extracted as described above. In both laboratories, Pb was determined by atomic absorption spectrophotometry. The Air and Industrial Hygiene Laboratory found 99% ± 2% of the Pb found by U.C. Berkeley. To evaluate the precision of the analytical method within the laboratory, a pool of dirt samples was homogenized and aliquots were analyzed on a daily basis. For fourteen determinations, the mean value was 6,488 ppm, with a coefficient of variation of 5.2%.

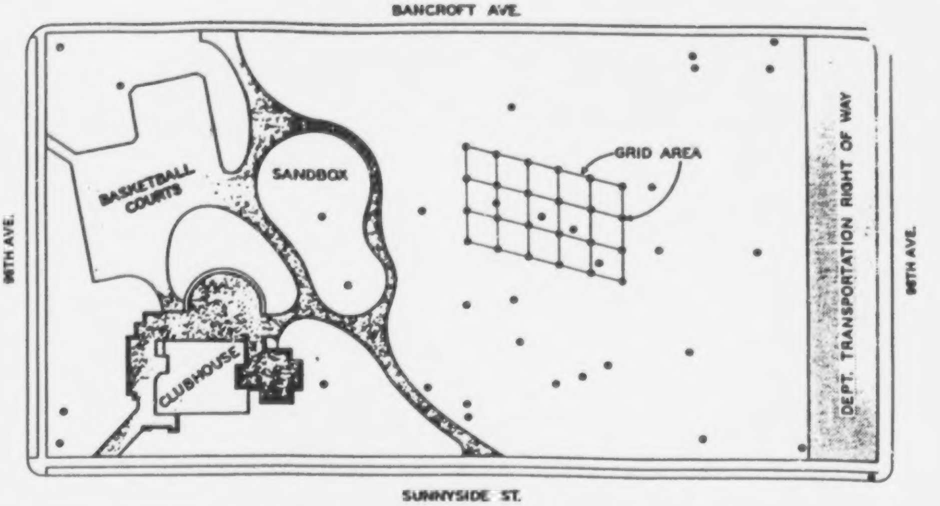
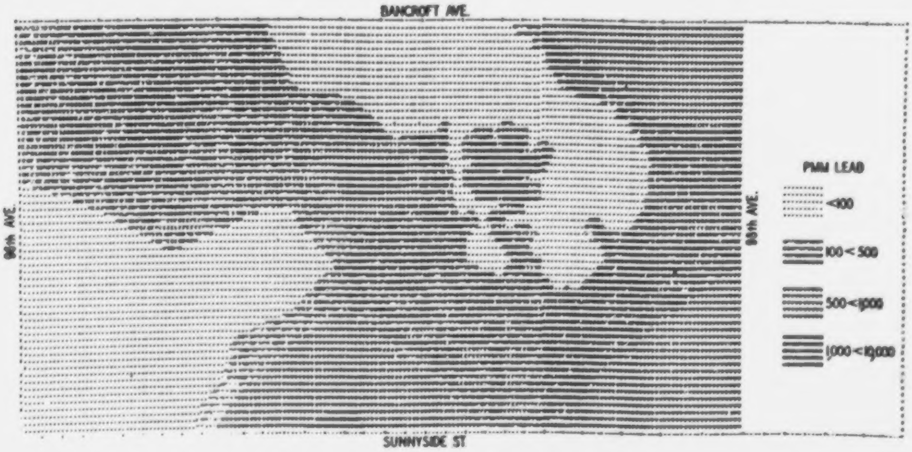

BANCROFT AVE.
BASKETBALL COURTS
SANDBOX
GRID AREA
CLUBHOUSE
DEPT. TRANSPORTATION RIGHT OF WAY
96TH AVE.
98TH AVE.
SUNNYSIDE ST.

Fig. 1. Schematic drawing of Verdese Carter Park, (● = sampling sites).

Lead contour maps were constructed for the surface, 15 cm and 30 cm horizontal planes using the U.C. Berkeley CDC 6400 computer and a computer program called SYMAP.[13] Sample point coordinates and associated Pb levels are input to the program which constructs a matrix containing the sample point values and interpolated values for areas between the sample points. Lead intervals were specified as: < 100, 100 < 500, 500 < 1,000, and > 1,000 ppm. The program prints out the matrix substituting a specified symbol (in this case, light to dark from low to high Pb level) for the data. The methods of interpolation and symbol definition are described elsewhere in detail.[14]


BANCROFT AVE.
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1,000 < 10,000
SUNNYSIDE ST

Fig. 2. Contour map of surface soil lead levels for the entire park.

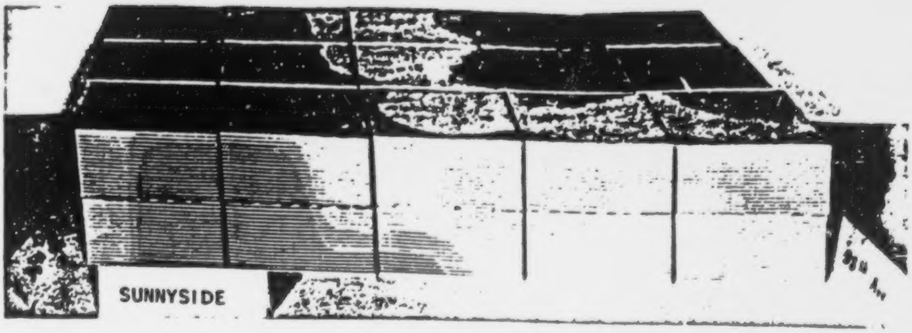

Fig. 3. Photograph of 3-D contour model.

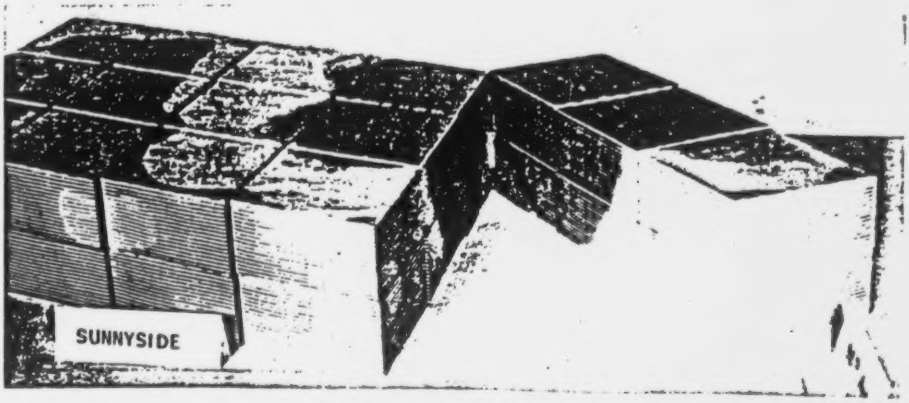

Fig. 4. Photograph of 3-D contour model.

Because each sample point yielded lead levels at the surface, 15, and 30 cm depths, it was also possible to interpolate vertical planes through the grid. The horizontal and vertical plane maps were pasted on styrofoam blocks to build the model of the grid area.

Results

Table 1 lists the range, mean, and median values for each depth for the 24-point grid area. The wide range of values from 13 to almost 100,000 ppm, and the median increase with depth makes it improbable that motor vehicle fallout is the primary source of contamination. Figure 2 is a contour map of the surface Pb in the park. Figures 3 and 4 are photographs of the three-dimensional model constructed for the grid area. The verticle profiles do not suggest vehicle fallout as the primary source. The figures demonstrate that the measurements at the three depths are consistent with one another, and with the assumption that the major source of contamination was the former battery plant. In order to test the validity of the model, six samples were subsequently collected in the grid area and the Pb concentrations compared with values predicted by the

Table 1.–Lead Concentration in Soil (ppm)

Depth	N	Range	Mean	Median
Surface	24	57 - 95,588	4270	198
15 cm	23	13 - 4234	374	203
30 cm	24	13 - 4546	1119	358

model. Five of the six samples had values which fell within the predicted or adjacent range.

Abatement and Restoration

Although there were a number of hazard reduction options available (e. g., covering the contaminated area with a barrier of concrete or asphalt) the most reasonable one, consistent with maintaining the original purpose of the park, was to remove the contaminated soil and replace it with uncontaminated soil. The model suggested that the pockets of heavy contamination were sufficiently close to one another that *selective* removal of soil in the area of concern was not a practical reduction procedure. Instead, all the soil had to be removed.

Before a decision was reached on the removal depth, a set of seven samples was collected from the surface to 90 cm from scattered locations. The results indicated a sharp reduction in contamination below the 30 cm depth. The mean and median values at approximately 90 cm were 68 ppm and 10 ppm, respectively. Thus, the decision was reached to remove all the soil within the contaminated area to a depth of 30 cm and replace it with 45 to 50 cm of clean soil. The contaminated soil was removed to a Class I sanitary land fill approved by the State Department of Health Services, Hazardous Materials Management Section. The new imported soil was tested for Pb by private laboratories and AIHL. The results were all low with AIHL values for six samples ranging from 15 ppm to 19 ppm.

Park development is now completed and the park is open to the public.

Biological Monitoring

In order to determine if neighborhood children who might have used the area prior to or during construction could have suffered undue lead burden, a voluntary screening program was set up in the neighborhood. In most cases children were first screened for erythrocyte protoporphyrin (EP), and following CDC guidlines, lead determinations were made only for those with EP $\geq$ 60 μg/dl. Measurement techniques are discussed in another report.[18] Two children of approximately 300 screened were found to have slightly elevated lead levels (31 and 32 μg/dl), but in neither case could it be established that the park was the source of the elevated level. These negative results are fortunate but not surprising, in view of the limited use of the area prior to and during construction, particularly by those most susceptible, i. e., children between the ages of 1 and 3 yr.

Comments

A potential public health lead hazard in soil in an urban park under development was recognized and eliminated using a strategy which included:

a) Awareness and concern by local residents of a possible problem.
b) Cooperative sampling and analysis of soil samples by county and state health departments.
c) A stepwise sampling strategy to identify the problem.
d) The presentation of experimental results in a three-dimensional computer model of the park to assist in developing the hazard reduction procedure.

One of the reasons this potential problem was recognized by local residents was the relatively short time between the destruction of the battery plant and the park development. This allowed for human memory to be aware of the transition in land use. This is not always the case, nor will it be in the future. In the many redeveloped projects underway today, millions of acres of land are being turned over or moved to new landfill locations. In some cases the soil may have large concentrations of toxic materials which could present a public health hazard in its new use. This illustrates three points: 1) more information is needed about the concentration of toxic materials in the land we live and work on; 2) a repository for this information, in usable form, is needed; and 3) there is a need for a method of transferring this information to future generations to compel those responsible for land changes to take action when necessary.

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The authors appreciate the efforts of A. del Rosario, G. Guirguis, G. Perez, Y. Yaffe, I. de la Fuente, E. Kothny of the Air and Industrial Hygiene Laboratory; H. Doner of U.C. Berkeley; and J. Glover and J. Winn of the Alameda County Health Care Services Agency. We are grateful to J. Rahman, a resident of Oakland who initially called our attention to the problem.

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Effect of Detergent on Protease-Induced Mortality and Pulmonary Histopathology

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ABSTRACT. Addition of an anionic detergent to aerosols of *Bacillus subtilis* protease markedly increased mortality in guinea pigs following a single aerosol exposure and altered the nature of the lung pathology after exposure. It was observed that the detergent inhibited in vitro activity of serum protease inhibitors and it is suggested that such impairment may be, in part, responsible for the observed in vivo effects of detergent on protease-induced mortality and pulmonary pathology.

INHALATION OF proteolytic enzymes can result in significant pulmonary disease. Allergic reactions following industrial exposure to trypsin, chymotrypsin, bromelin, and papain have been reported[1-3] in which asthmatic responses were the predominant clinical feature. Experimentally, aerosol or intratracheal administration of papain can induce emphysematous lesions in the lungs of hamsters.[4,5] Similar lesions can be produced by administration of lysosomal enzyme preparations from either polymorphonuclear leukocytes[6] or alveolar macrophages,[7] suggesting that release of endogenous enzymes from inflammatory cells subsequent to their recruitment into the lung may be involved in the pathogenesis of emphysema.

Following reports of a high prevalence of allergic pulmonary disease in workers exposed to bacterial proteases in the course of manufacturing commercial enzyme detergents,[8] the authors conducted experiments to determine the effect of detergent on the pulmonary response to aerosolized protease. We observed that a single aerosol exposure of guinea pigs resulted in mortality and pulmonary pathology and that addition of an anionic detergent into aerosols of the enzyme increased the clinical and pathologic response to the protease.[9]

It is the purpose of this report to describe the response of guinea pigs to aerosolized *Bacillus subtilis* protease and to assess the role of detergent in these reactions.

Methods

Female Hartley strain guinea pigs (Connaught Labs, Toronto) weighing 300 to 350 g were used in all experiments.

B. subtilis endopeptidase (Subtilopeptidase A [SPA])

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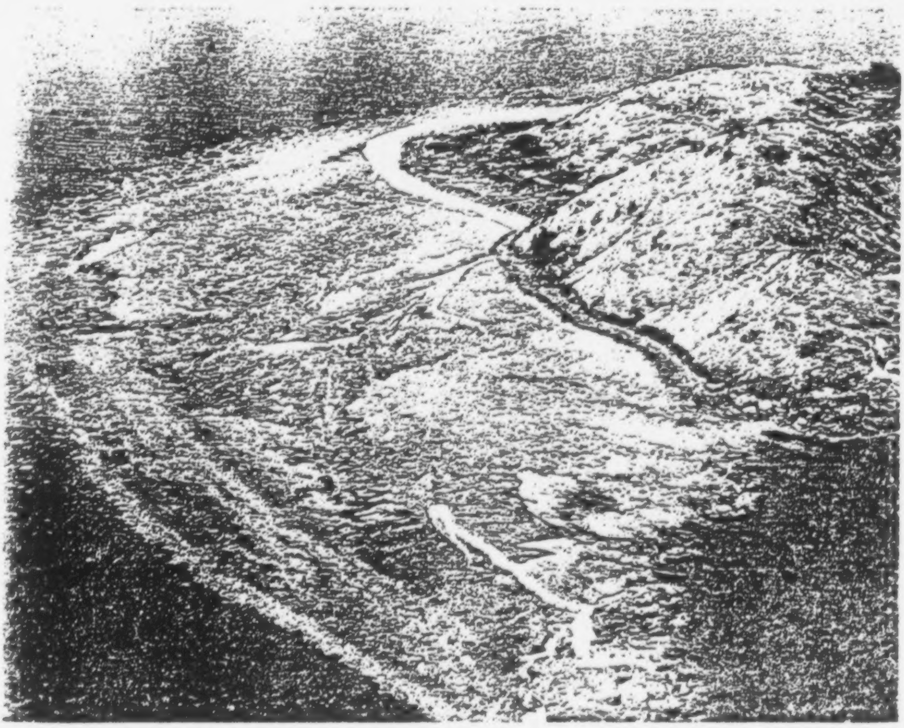
Science, Technology and Society

ANZAAS
Vol. 8 No. 3 March, 1977



Holbourne Island (Micro-atolls, p 79)

25-77

Registered for posting as a periodical — Category B

research reports

Contamination of Soils and Vegetables near the Lead-zinc Smelter, Port Pirie, by Cadmium, Lead and Zinc

K.G. TILLER and M.P.C. de VRIES*

The contamination of home gardens within the city of Port Pirie may lead to above normal intakes of heavy metals by a few householders because of increased metal contents of some vegetables. A greater risk may arise from the eating of contaminated soil by some small children. This work extends earlier studies (Tiller *et al.*, 1975, and related papers) which emphasized the need for investigation of contamination in the Port Pirie area to quantify the contamination and to consider some of its implications for human health.

Vegetables in season and corresponding soils (0-10 cm layer) were sampled from most residential areas of Port Pirie: about 5% of the home gardens were sampled. Cucurbit leaves were collected to assess current fallout. The approach of Little (1973) was followed; leaves were split along each side of the mid-rib with one half being rinsed with dilute acid (0.04% HNO_3) and the other not. The procedure was checked on cucurbit leaves from uncontaminated areas. Acid rinsing commonly removed appreciable amounts of lead but only small amounts of cadmium and zinc Soil samples were extracted for 7 days with 0.1 M EDTA at pH 6 and extracts analysed by atomic absorption spectrophotometry (A.A.S.) with matrix correction. Plant material was analysed by the method of de Vries *et al.* (1975b)

The concentration of lead in urban soils is shown in Figure 1. The distribution of zinc and cadmium is similar to that of lead. The main soil pollution is to the SSE of the smelter, in agreement with the regional trend shown previously (Tiller *et al.*. 1975). Soil contamination decreased markedly over a short distance beyond the southern limits of the city where metal contents of wheat grain had previously been shown to decrease sharply. There were also marked variations in soil composition superimposed on the general trend. These are due to the relatively recent use of uncontaminated garden filling, especially in the NW sector, and past applications of smelter slag. We found large ranges of extractable metal contents (μg/g surface soil) in home gardens, viz., Cd (0.03 – 14), Pb (4 – 2100) and Zn (2 – 1500). Our EDTA extraction proceedures removed about 85% of cadmium and lead but only about 50% of total zinc content from contaminated soils. The highest cadmium contents, in particular, were not always in the main areas of contamination. Thus the degree of contamination of an individual garden cannot be reliably estimated from its location.

Comparisons of metal concentrations of different vegetables (Table) confirmed the well-known observation (UK, 1972, 1973) that root and leaf vegetables accumulate cadmium and lead. Carrots had the highest cadmium concentration. Tomatoes had low metal levels. Ranges of metal concentrations in all samples appreciably exceeded those of the reference Adelaide samples for cadmium and lead and were similar to those quoted by Beavington (1975) for Wollongong, NSW.

The geographical distribution (Figure 2) of zinc concentrations in the acid-rinsed cucurbit leaves, indicating the uptake by the roots, corresponded closely with that of zinc in soils. A Similar pattern was found for cadmium. Surface contamination (loss on acid-rinsing) contributed up to two-thirds of the Zn content of cucurbit leaves, especially in the north and north-east sectors. This contamination was not restricted to areas where soil had a high zinc content and probably results from smelter fume. This effect would have been reduced by the predominantly southerly winds prior to sampling.

Up to 80% of the lead associated with tomatoes grown about 1 km SW of the smelter, some on uncontaminated soil filling, also occurred as surface contamination. These examples highlight the possibility of surface contamination of garden produce and emphasize the need for good hygiene.

None of the metals in the vegetables analysed exceeded the lead and zinc standards prescribed by the South Australian regulations (SA, 1964). However, the weekly tolerable intake of 3000 μg Pb and 400-500 μg Cd for adults, as recommended by FAO (1973), could be exceeded by some householders from vegetables alone

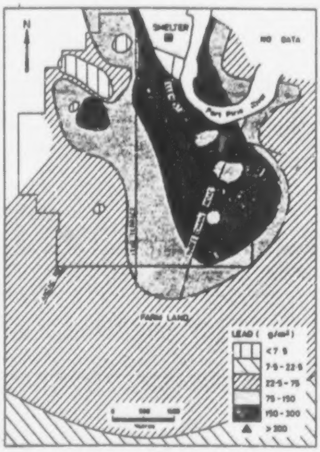
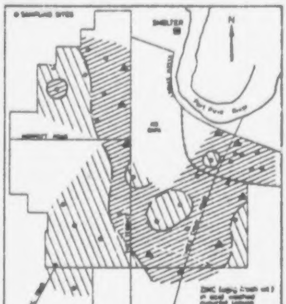


FIGURE 1 (left)

The accumulation of lead in Port Pirie soils

FIGURE 2 (below)

The zinc concentration in acid washed cucurbit leaves.



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* CSIRO, Division of Soils
Glen Osmond, South Australia

Vegetable	No. Samples	Pb	Cd	Ni	Cu	Zn	Mn
Silver beet	26	0.22 – 2.2	0.04 – 0.49	0.02 – 0.14	0.71 – 2.1	4 – 29	2.5 – 56
Tomatoes	44	< 0.01 – 0.07	0.01 – 0.21	0.01 – 0.27	0.60 – 1.10	1 – 10	0.6 – 1.3
Carrots	21	0.09 – 1.6	0.02 – 2.6	0.02 – 0.33	0.51 – 1.3	3 – 8	0.5 – 2.8
Rhubarb	12	0.04 – 0.74	0.02 – 0.19	0.02 – 0.31	0.23 – 0.79	2 – 4	0.1 – 5.3
Beetroot	5	0.14 – 1.9	0.03 – 0.36	0.02 – 0.12	0.87 – 2.7	6 – 31	1.5 – 10
Capsicum	6	0.05 – 0.12	0.02 – 0.12	0.01 – 0.16	0.78 – 1.2	2 – 4	0.7 – 1.4
Miscell. (6 varieties)	11	0.03 – 0.33	0.01 – 0.18	0.02 – 0.16	0.32 – 2.3	3 – 19	0.4 – 8.8
Overall Range Pt. Pirie Study	125	< 0.01 – 2.2 (0.55)	0.01 – 2.6 (0.14)	0.01 – 0.33 (0.05)	0.23 – 2.7 (1.0)	1 – 31 (7)	0.4 – 55 (3.0)
Overall Range* Adelaide samples	98†	0.002 – 0.34 (0.08)	0.001 – 0.29 (0.02)	0.003 – 0.3 [illegible]	0.11 – 5.0 (0.9)	0.6 – 85 (4)	0.5 – 180 (2.5)

TABLE

Summary of metal contents of vegetables from Port Pirie home gardens (μg/g fresh weight)

*from Tiller *et al.* 1976

† Ni values are based on 18 samples only.

75 percentile values are given in brackets.

during the growing season. The possible cadmium intake from the vegetables grown in one home garden that was sampled was 5 times greater than the FAO limit (assuming a daily consumption of 300g of these vegetables).

Apart from their contribution to the composition of plants, contaminated soils deserve attention as sources of heavy metals in the human diet. The common ingestion of soil and other non-food material by small children (N.A.S., 1972), referred to as pica, raises the possibility of a more insidious health risk. The average daily intake of 130 μg Pb for a 10 kg child (FAO, 1973) would be exceeded four times by the daily consumption of one gramme of soil from the upper third of the gardens sampled. In several gardens, the corresponding increase in lead intake would be at least 10 times. Lead intake by children with pica raises concern because of the difficulties in recognising subclinical lead poisoning in children and the possible effects of early exposures to lead on their health and early development and behaviour in later life (N.A.S., 1972; Bryce-Smith and Waldron, 1974; de la Burde and Choate, 1975).

Hazards arising from the consumption of vegetable produce with elevated concentrations of metals cannot be adequately assessed without a complete knowledge of the local diet, the contribution of metals from non-food sources and more reliable data on the levels of metals which affect human health in the long term. It is clear, however, that some householders relying on home-grown produce may exceed recommended intakes of cadmium and, to a lesser extent, lead. Of greater concern is the potential for substantially increased intakes of heavy metals by some young children who eat contaminated soil.

The analyses were made by L.R. Spouncer and B. Zarcinas. The cooperation of the South Australian Department of Public Health in assisting with sample collection is gratefully acknowledged.

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Coral Micro - Atolls, Tropical Cyclones and Reef Flat Morphology: A North Queensland Example

DAVID HOPLEY
PETER ISDALE*

Micro-atolls are flattened disc-shaped coral colonies located on reef flats, and consisting of living, sometimes raised coral rims and dead algal covered centres. The majority are formed from the massive rounded structures which develop from evenly growing hemispherical coral colonies whose upward growth is interrupted by subaerial exposure. Upward growth appears limited to no more than 5 cm above the low-water level, a height at which complete desiccation of the upper part of the colony is prevented by the lapping of small waves, protective mucus coatings and capillary action. However, particularly in areas of large tidal range, it is clear that the sub-horizontal upper surface of the micro-atolls is not related to an open water low-tide level which varies from day to day, but to a consistently achieved moated level generally above all but the neap low tides.

Moating may take place in a number of situations. Most commonly the damming agent is a ridge or rampart of *Acropora* shingle, the formation of which along the Great Barrier Reef appears associated with high-energy events, usually tropical cyclones (see Moorhouse, 1933, for example). However, whilst this coarse, loosely packed material may retard drainage at low tide, it becomes a more efficient dam when, in time, its basal portions become compacted and cemented. Sometimes the uncemented upper portions of the ridge may be removed, and the cemented basal parts in the form of 'basset edges' (Spender, 1930, Steers, 1929) alone become the moating agent. Elsewhere, particularly where the tidal range exceeds 3 m, the algal rim of the reef may form the dam, the calcareous algae (*Porolithon* and *Lithothamnion*) growing to levels exceeding those of coral growth.

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Report

TENTATIVE GUIDELINES FOR ACCEPTABLE CONCENTRATIONS OF CONTAMINANTS IN SOILS *

BY

M A SMITH

BUILDING RESEARCH STATION
DEPARTMENT OF THE ENVIRONMENT

Summary

The aim of this paper is to provide guidance that would give an adequate degree of protection against the potential hazards caused by the presence of toxic elements in soils found on contaminated land. It attempts to define concentrations which, if not exceeded, can be regarded as "acceptable" as presenting no significant hazard. The suggested acceptable concentrations are presented in Table 1. They take account of problems posed by uptake of food crops, phytotoxicity and ingestion of contaminated soil.

Before using the guidelines to assess the suitability of a particular site, it is essential that an adequate site investigation is carried out. If then the observed concentrations are within the acceptable values, development can proceed without any remedial action being required. However, if any values are exceeded consideration should be given to the necessity for remedial or ameliorative action. On this basis, concentrations exceeding the guideline values are not therefore necessarily 'unacceptable', although they may be 'undesirable'.

APRIL 1981

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* NOTE: This paper was produced for presentation at the Geological Society Discussion Meeting "The Hazards of Contaminated Land", held in London on 15 May 1981. It is based on previous consultation papers of the Inter-departmental Committee on the Redevelopment of Contaminated Land (ICRCL), and should be read in conjunction with the current version of the full text (ICRCL 38/80) which is available from the Committee at the following address : Room S10/16, 2 Marsham St, London SW1P 3EB. Further information about ICRCL can also be obtained from this address.

1. INTRODUCTION

1.1 The increasing need to redevelop land previously used for industrial purposes, particularly in inner cities and urban areas, gave rise to a requirement for advice on the problem of contamination and other difficulties. It was recognised that such advice should be provided by Central Government, and this led to the establishment of an Inter-departmental Committee (ICRCL) to co-ordinate the different specialist viewpoints from which the advice was drawn. The secretariat for ICRCL has been provided by staff of the Central Directorate on Environmental Pollution (CDEP) within the Department of the Environment. On behalf of ICRCL, CDEP provides advice on contaminated land problems to local and regional authorities who have the executive responsibility for action. This advice is particularly concerned with the assessment of the effects on health and the environment of toxic substances, including heavy metals. Guidance on the measures needed to enable contaminated land to be redeveloped safety and acceptably is one of the most important problems.

1.2 The aims of UK environmental policy are:

(i) to protect human life and health,

(ii) to safeguard plants, animals and other environmental resources of actual or potential use to man,

(iii) to minimise permanent adverse effects on the natural and man-made environment,

and

(iv) progressively to improve the general state of the UK environment.

Although to some extent all of these aims are relevant to the subject of contaminated land, the most important for present purposes is the first.

1.3 Problems occur principally when contaminants are present in concentrations potentially capable of affecting public health. The primary hazards for public health in relation to contaminated land are:

(i) uptake by food crops of elements toxic to man,

(ii) ingestion (or in some cases, inhalation) of soil,

(iii) dermal effects (eg. contact with skin irritants such as chromium).

In addition, for some purposes the presence of contaminants in concentrations likely to be toxic to plants is also relevant. Which of these hazards will be the most important on a particular site depends on the type of land and the form of contamination present. The most acute problem for metal uptake by food crops is on allotments or large gardens, whereas for domestic gardens direct ingestion of soil by children may be more important. But whichever hazard is the most important, it is usually necessary to determine those concentrations of contaminants which will be significant in the particular circumstances.

1.4 It was therefore decided by ICRCL to produce guidance on acceptable concentrations of contaminants in soils found on contaminated land (1). The approach adopted was to try to identify concentrations which, if not exceeded on a particular site, could be regarded as presenting no significant hazard: such concentrations were termed "acceptable". If these "acceptable" values are exceeded, it follows that remedial or ameliorative measures may be needed, depending on the particular end use proposed for the land: the fact that the acceptable concentrations are exceeded may be undesirable, but not necessarily unacceptable.

1.5 At still higher concentrations, the presence of contamination would make it essential to carry out remedial measures appropriate to the indended use: such concentrations are thus, by definition, "unacceptable". It is however difficult to specify concentrations of contaminants which should not in any circumstances be exceeded. The purpose of the present paper is to suggest values which could be regarded as the lower limits, or "trigger" concentrations, *at which the need for action should be considered*. The main feature of the paper is Table 1, which contains the suggested concentrations for various possible end uses of contaminated land.

1.6 In this paper, use has been made of certain existing guidelines and regulations in seeking to define guidelines for contaminated land, rather than attempting to work from first principles. Consequently the safety factors built into the limits suggested in Table 1 for different elements are unlikely to be comparable. The hazards presented by individual elements are not discussed in this paper, which has been prepared to serve as a basis for discussion of the principles and suggested limits. The paper does not attempt to deal with the complete range of topics which may be relevant to a full assessment of such sites. Copies of an ICRCL paper (2), which discusses these topics in detail, can be obtained from CDEP. Some of these points will however be discussed by later speakers today.

2. GUIDELINES FOR TOXIC SUBSTANCES

2.1 *Their role and function*

Guidance is required by people with a wide range of backgrounds, including environmental health, planning, and the professions involved in building and construction. For this reason the guidelines must be presented in such a way as to facilitate recognition of potential problems. Once a problem has been identified, following an adequate investigation of the site concerned, the guidelines may require further interpretation by specialist advisers working in association with members of the development team. The guidelines must therefore assume the exercise of 'professional judgement' on the basis of personal experience and knowledge. For these more detailed applications, they need to take account of the practical and economic implications (in terms of the cost of remedial works), and the concentrations which are commonly encountered in rural and urban soils.

2.2 Restrictions

The guidelines proposed in this paper are intended to apply only to development of contaminated sites. They do not imply that similar conditions should be created voluntarily by importing contaminated material into a clean area, nor do they indicate the standard which should be achieved when ameliorative measures are undertaken. They should not be applied to an existing development which is found to be on contaminated ground without careful consideration. There is a distinction between creating a new situation and dealing with existing hazards. For some elements (eg. zinc) the need to have viable plant growth requires limits to be set below those at which a hazard to human health is likely to exist. Whilst this paper is concerned with hazards due to excesses of certain elements being present in the soil, trace quantities of some of the elements in question can be vital to life. Deficiencies, or imbalances between elements, can equally lead to adverse effects upon the health of man, animals and plants.

2.3 Types of land use

The guidelines concentrate on those uses of land likely to be of concern following comprehensive physical development: allotments, 'large' and 'small' domestic gardens, amenity areas, recreational and educational land, and public open space. Not all land falls conveniently into these categories and there will often be special circumstances to take into account, for example how to treat areas of amenity grass and public open space within commercial and industrial developments.

The idea of dividing land-use into separate categories with different guideline limits has been criticised, because land use can change quite rapidly. One view is to say that for each contaminant there should be a single guideline for all land irrespective of use. The more common position is that, as distinctions for acceptable values are drawn for 'small' and 'large' gardens for only a few elements, this division could be dispensed with. Further views on this question would be useful.

2.4 Use of the guidelines

When a particular site is assessed in relation to the guidelines each contaminant should first be considered individually. The pattern and extent of contamination for all contaminants should then be evaluated. There may be instances where although the acceptable values are not exceeded for any individual element, concentrations of a number of elements are enhanced and it is consequently prudent to take ameliorative action. To aid the application of the guidelines in practice, it is suggested that, they should be stated in the form:

> "No individual 'spot' sample taken from the top 450mm should exceed the acceptable concentration and there should only be an acceptably low probability (say 1 in 100) that any significant proportion of soil exceeds the limit".

The level of probability should be chosen according to the particul circumstances, and should not imply such a rigorous standard as would be impossible to achieve under realistic conditions.

In practice it will rarely be possible to obtain sufficient data for a statistical assessment to be made and reliance must instead be placed on the judgement of the specialist advisers (eg. analyst, environmental health specialist, project engineers) in assessing the analytical data and whatever other information may be available.

TABLE 1

Top 1¾ inch of soil

TENTATIVE GUIDELINES FOR ACCEPTABLE CONCENTRATIONS OF SELECTED ELEMENTS IN SOILS WHERE NEW DEVELOPMENT OR REDEVELOPMENT IS TAKING PLACE

ELEMENT	'SMALL' GARDENS (Note f)	'LARGE' GARDENS & ALLOTMENTS (Note c)	AMENITY GRASS (Notes a & c)	PUBLIC OPEN SPACE (Note b)
	'ACCEPTABLE' CONCENTRATIONS (mg/kg dry soil- Note c)			
CADMIUM	5	3	12	15
LEAD	550	550	1500	2000
MERCURY	1.5	1	4	20
CHROMIUM-TOTAL	600	600	1000	1000
CHROMIUM - HEXAVALENT	25 soluble	25 soluble	25 soluble	25 soluble
ARSENIC	20	10	40	40
SELENIUM	3	3	3	6
BORON (Note d)	3	3	6	6
BARIUM	125 soluble	125 soluble	125 soluble	1000
ANTIMONY	60 soluble	60 soluble	60 soluble	500
FLUORINE	800	800	800	1000
	PHYTOTOXIC GUIDELINES 'Acceptable' or 'Trigger' Concentrations			
ZINC		130)		
COPPER		50)'available' soluble in 0.05M EDTA		
NICKEL		20) (note e)		
	Maximum Normally Tolerable Concentrations			
ZINC)	280	280	280-560	280-560)
COPPER)	140	140	140-280	140-280)'available'
NICKEL)	35	35	35-70	35-70) (note e)

NOTES:

a. Includes schools, play areas, and recreational areas, around residential development, where there may be regular contact by small children. Areas where there may be more intensive use by small children (eg nursery schools) should be treated as domestic gardens.

b. Includes formal playing field areas, parkland and informal recreational areas.

c. (i) Unless specified otherwise, all values are total concentrations on spot (ii) 'Soluble' determinations in 0.1M HCl or solution corrected to pH 1.0 if alkaline substances present. (iii) 'Available' determination by standard ADAS analytical methods.

d. Water-soluble boron by standard ADAS method.

e. It is commonly assumed that the effects of zinc, copper and nickel may be additive. The zinc equivalent concept may be helpful in evaluating potential toxicity where all three are present. Comparative toxicities may differ from those implied by the suggested limits. The higher end of the range is applicable to calcareous soils.

f. Small gardens arbitrarily defined as less than 75m^2 in area (see section 3.2)

g. It is assumed that the soil pH is about 6.5 and that attempts will be made to maintain this value. If the pH is lower than this, toxic effects and uptake will be enhanced for most elements.

2.5 Defining "acceptable" concentrations

The form and degree of contamination encountered vary widely from site to site. Once a hazard has been recognised, an assessment is made of whether the risk is significant. The type of assessment depends upon the problem and the information available.

The main difficulty in identifying hazards and risks is the problem of detecting effects. Toxic elements are rarely present in humans in quantities which produce overt clinical symptoms and those effects which are discernible, for example changes in blood enzymes, are difficult to interpret. Thus, in attempting to set guidelines for acceptable concentrations of toxic elements in soils, it is at present necessary to incorporate safety factors which are as much a matter for judgement as for scientific assessment. They should therefore to some extent take into account the industrial, economic or social importance of the activities giving rise to the hazard. Within this constraint the safety factors are based on such experience and experiment as is available. They must be regarded in a sense as arbitrary, but also as the judgements of a prudent person. There is no common "yardstick" which is applicable to all sites. Each hazard requires the choice of a "yardstick" most appropriate in the particular context. The only "absolute" criteria would be produced by "unacceptable" concentrations. Such conditions would be recognised as being so intolerable that the need for action is obvious. Conversely there are other cases where no action at all is required. The problem is what lies in between.

One possible approach is that conditions should not become more hazardous than is usual or normal, thus avoiding a rise in mortality or morbidity: this is perhaps the method that is most applicable in the present context. Some other principles which have been used in analogous contexts are:

> (i) With the disposal of wastes to land, those that are controlled are subject to a two tier approach: there are general restrictions in Part 1 of the Control of Pollution Act 1974, but individual site licences take account of local conditions.
>
> (ii) For substances such as nitrates in water, a three-level approach operates: nitrate concentrations being regarded as (a) unacceptable, (b) acceptable or (c) desirable.

For contaminated land problems, the approach which has been adopted so far by ICRCL resembles that outlined above for nitrates in water, in that there is a similar, but not identical, triple distinction: (a) unacceptable (must do something); (b) undesirable (should consider doing something); (c) acceptable (need not do anything). This paper deals only with acceptable limits, but comments on this reservation and views on appropriate limits for the first two purposes are invited.

3. DERIVATION AND APPLICATIONS OF ACCEPTABLE CONCENTRATIONS

3.1 Derivation

The longstanding ADAS guidelines for agricultural and horticultural land, which have been incorporated in the guidelines for the disposal of sewage sludge to land (3) are based on the concept of 'plant available' metals - in particular zinc, copper and nickel, but this concept presents serious difficulties. Availability to plants depends upon many factors including pH, the content of organic matter, redox potential, macronutrient status and cation capacity of the soil. Whilst some of these factors are, and need for good commercial reasons to be controlled in agriculture and horticulture, there can be no certainty that this will be done elsewhere. Thus some allowance must be made for possible changes of availability with time. In addition, the difficulty of selecting an appropriate extractant to determine availability must be considered.

The interim guidelines for the disposal of sewage sludge to land indicate the quantities of toxic elements which could be added to a normal soil over a 30 year period, but this does not allow for soil which was already contaminated for one reason or another. Consequently, future guidelines are likely to be based on the acceptable total quantity which may be reached after 30 years. The idea of a gradual build-up to the limit is a considerable improvement since it enables any adverse effects on crop yield to be detected and allows adjustments to be made in the proposed limits in the light of new knowledge.

The sludge guidelines, from which those given in Table 1 are derived, are based on the assumption that the pH of the soil will be maintained at about 6.5 for arable soils and 6.0 for grassland. However, soil pH values of less than 6 are common for woodlands, heathlands and permanent grasslands, except on chalk or limestone. Many contaminated sites will have pH values outside the expected normal range: extremes of acidity and alkalinity may be encountered. As the uptake of metals by plants tends to increase as the acidity increases (molybdenum, selenium and hexavalent chromium are exceptions to this general rule as uptake increases with increased alkalinity) the safety factors assumed in the sludge guidelines, both in respect of toxicity to plants and uptake by food crops, may not be applicable unless steps are taken to control the pH of the soil. This may be an essential step in any remedial treatment of the site. Moreover, there is a natural tendency for soils to become acid under certain environmental conditions (eg. permanent pasture, woodland, acid rainfall as found in urban areas), and maintenance of the pH at the assumed values cannot be guaranteed.

3.2 Uptake of metals by crop plants

Different acceptable concentrations are proposed in Table 1 for certain elements for 'small' and 'large' gardens, the latter being grouped with allotments. This distinction is considered to be justified for elements where the concern is predominantly for uptake by food crops (eg. cadmium), but not where possible ingestion of soil is considered to be the primary hazard (eg. lead).

Most gardens in modern developments are likely to be 'small' in the sense that great determination would be required to obtain a substantial proportion of total vegetable and fruit intakes from them. Other gardens are large enough to contain a substantial vegetable plot or soft fruit area. Any division between 'large' and 'small' will be arbitrary, but for the purposes of Table 1 gardens of less than $75m^2$ are considered small (a half-size allotment is about $125m^2$). The Table also assumes that in smaller gardens there will be a tendency to concentrate on high-value and salad crops, whereas in a larger plot a substantial area would in addition be set aside for potatoes and brassicas. Certain salad crops (eg. lettuce, radish) tend to accumulate toxic metals, so that a small area devoted to such crops could sometimes lead to a greater degree of exposure than a larger area devoted solely to brassicas and potatoes.

3.3 Phytotoxicity

Phytotoxicity is of practical concern, since soil which will not support adequate plant life cannot be considered as suitable for domestic gardens, amenity areas etc. A distinction should be made between areas where continuing health of plants on a long-term basis is highly desirable, such as domestic gardens, and those where, whilst loss of plant cover is undesirable, it is easier to rectify any failures or deficiencies, such as landscaped areas around commercial or industrial development.

Grass is more resistant to zinc, copper and nickel than many other plants. Consequently, as shown in Table 1, higher concentrations of these and other toxic elements are acceptable for areas set to permanent grass, such as amenity and parkland grass. For domestic lawns, which might be dug up at some later date and replaced by ornamental or food-crop plants, lower concentrations are needed.

3.4 Ingestion

The possible hazards from the ingestion of toxic metals by children from toys and graphic materials have led to the specification of limits for various elements in The Toys (Safety) Regulations 1974 and The Pencils and Graphic Instruments (Safety) Regulations 1974 (4, 5). The limits are based on determinations of acid-soluble metals, to assess the 'availability' of the metals to a child should the metals be ingested. Direct extrapolation from these Regulations is unlikely to be a satisfactory basis for specifying acceptable values in soils, particularly where the acceptable level is defined in terms of solubility in dilute HCL. Very little is known about the response of soils and soil-like materials in the type of test specified by the Regulations, and the methods might not be appropriate for certain types of soil - particularly calcareous soils (ie. those containing calcium carbonate) which might react with, and consequently neutralise, the acid used in the test.

In Table 1, limits based on acid-soluble concentrations have been used for the time being for certain elements until revised limits based on total concentrations are ready. The suggested values have been reduced, in order to allow for the greater volume of material which might be ingested. The extent to which they should take account of children with a tendency to 'pica' (the voluntary ingestion of non-food objects), as opposed to the child who merely has dirty hands or drops his sweets on the ground, is debateable.

3.5 Skin Contact

Skin contact with contaminants is a potential hazard during construction and reclamation work, and may also be of concern for site investigation teams and the eventual users or occupants of the site. Potential hazards to workers can be assessed from the results of site investigations, and safe methods of working adopted to minimise any risk. Normal industrial hygiene standards would apply and the Health and Safety Executive should be consulted as necessary. Protection of workers who might come onto the site at some later date presents greater difficulties. No reliance can be placed on a particular group of workers being aware of the potential problems many years in the future. Consequently any remedial treatment carried out should at least provide a measure of protection to workers engaged in standard maintenance work eg. repair of telephone wires, gas and water pipes.

The assessment of long-term risks, particularly to occupants or users, is difficult. The limits set out in Table 1 for other purposes would probably offer sufficient protection and no specific allowance for protection against dermal effects has been made.

4. REFERENCES

1. Redevelopment of Contaminated Land: Acceptable Levels of Contaminants in Soils (Consultation Paper). DOE Central Directorate on Environmental Pollution, London, 1979; ICRCL 16/78(6).

2. Redevelopment of Contaminated Land: Tentative Guidelines for Acceptable Levels of Selected Elements in Soils. DOE Central Directorate on Environmental Pollution, London, March 1980; ICRCL 38/80.

3. Report of the Working Party on the Disposal of Sewage Sludge to Land (Department of the Environment and National Water Council, London, 1977).

4. The Toys (Safety) Regulations 1974, Statutory Instrument No. 1367, HMSO 1974.

5. The Pencils and Graphic Instruments (Safety) Regulations 1974, Statutory Instrument No. 226 (HMSO 1974).

IV. Memos/Reports Showing Chronological Review of QA/QC Procedures

QA 1

(108)

Ex. 3

Lockheed Engineering and Management Services Company, Inc.

Environmental Programs Office
P. O. Box 15027, 944 E. Harmon Avenue
Las Vegas, Nevada 89114 (702) 798-2175

October 30, 1981

Environmental Protection Agency
P.O. Box 15027
Las Vegas, Nevada 89114

Attention: J. Gareth Pearson

Subject: Review of Dallas Study on Lead and Cadmium for Analytical Merit and Quality Assurance; (EPA Contract #ot-0918-NTEX)

Stephen J. Simon
Principal Scientist
Quality Assurance Section

SJS:jk

cc: T. A. Hinners
W. D. Reynolds
J. O. 74.03

REVIEW OF DALLAS STUDY ON LEAD AND CADMIUM FOR ANALYTICAL MERIT AND QUALITY ASSURANCE.

Summary

In general the Dallas Lead/Cadmium study represented a fair first effort in terms of analysis technique and quality assurance. Given the level of effort awarded (~ $9,000.00) it should not be construed as a final word to base precedents or possible long-range policy decisions on. The analytical and quality assurance reported had several serious deficiencies which perhaps would be clarified if the raw analytical data and actual quality assurance data was included.

Contanimation Control Procedures, Storage and Sample Custody

Adequate contamination control procedures were reported for tap water and housedust sampling and all of the sample preparation techniques. Sample, storage, and preservation techniques used were within EPA recommended procedures. However, no mention was made of contamination control methods for soil sampling - such as precleaning the coring device and whether or not it was cleaned out after each sample. Additionally any chain of custody protocols for samples was not mentioned although it appeared not to be considered pertinent for this study since legal followup was unlikely.

Sampling Protocol

Ken Brown's and Tom Hinners' comments adequately covered the many deficiencies in this area and it is evident that the study could only be a very preliminary one at best due to problems with the site selections, number of samples per area, lack of composites, depth of sampling, wind direction, and other factors to discern if the lead found is actually due to the smelting activities or is background from automobile exhaust.

Sample Preparation

Soil

Soil samples were dried, ground and sieved (250 micron sieve) and five grams weighed for analysis. Some mixing would naturally occur during the grinding and sieving operations but no mention was made of consciously making a homogeneous sample for analysis or how the 5 gram analysis portion was taken (grab, cone/quarter, etc). Likewise no mention of replicate samples (see QA comments) from a given soil sample was mentioned to check the sample homogenity. What was the total sample weight? While a 5 gram portion for extraction is sizeable, is it truly representative of a core sample particularly without a deliberate blending step?

The extraction step is adequate and can be referenced in the literature (report should have included a reference). Data should be noted as "Extractable Pb and Cd". Total Pb and Cd would imply complete sample dissolution. No effort was implied or likely intended at metal speciation.

Dust Trays

Sample workup consisted of a vigorous HNO_3 extraction - again "Extracted Pb and Cd". Entire sample was analyzed.

Dust Wipes

Towlette extracted for Pb and Cd - Extractable Pb and Cd. No mention was made of extracting and analyzing a "blank" towelett for residual Pb and Cd.

Water Samples

Fairly standard EPA procedure. No problems detected.

Data

All data was reported to one decimal point rather than by number of significant figures (three maximum would have been more reasonable).

Quality Assurance

To quality assurance procedures mentioned in the report consititute a bare minimum for an analytical study.

1. The study mentions that QA included "the use of reagnet blanks," but doesn't elaborate to say if a blank dust tray or towlette was analyzed or if the reagent blanks were indeed significantly high enough to correct the sample data. As mentioned above, however, the contamination control procedures used (cleaning, high purity acids, analyst hygiene, etc.) were documented and appear adequate to prevent the main known sources of sample contamination.
2. The study mentions that QA included the "determination of a recovery factor for each type of extraction", but did not document what these factors were and if indeed they were used to correct the data to a 100% extraction.
3. The study mentions that QA included "replication of 10 percent of all AAS readings" but does not elaborate. This can be construed to mean a single AAS reading for all but 10% of the samples which is inadequate or that 10% of the samples were analyzed in replicate which is more in line with current QA/QC requirements. In any AAS measurement where meaningful, defensible results are expected the sample should at a minimum be read in duplicate.
4. The study mentions maintenance of the AAS linear analysis region by using a calibration curve, monitoring the standard solutions every tenth sample at a minimum and the diluting of samples outside the linear concentration

region.

The study does not mention in line with the above the following important QA/QC considerations.

a. The source of analytical standards for calibration.

b. Whether the calibration was verified with NBS traceable reference solution.

c. No precision data was included. AAS precision or replicate sample data.

d. No accuracy statement was made. A standard reference soil should have been analyzed with the soil samples to help verify the entire analytical scheme.

It would have been worthwhile if the author's would have consulted an analytical chemist skilled in environmental measurements and QA/QC prior to embarking on this contract. Such a consultation most likely would have improved the data reliability.

95
ΦA 2

U.S. ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF RESEARCH AND DEVELOPMENT
ENVIRONMENTAL MONITORING SYSTEMS LABORATORY-LAS VEGAS
P.O. BOX 15027, LAS VEGAS, NEVADA 89114 • 702/798-2100 (FTS 545-2100)

Date: November 24, 1981

Reply to Attn of: EAD

Subject: Dallas Lead Study

To: The Files

Enclosed is a summary of notes taken by Gareth Pearson and edited by James Mullins concerning the Dallas Lead Study. The information in the notes was relayed by telephone to Lamar Miller on 10/29/81.

James W. Mullins
for E. Paul Richitt, Jr.
Director
Exposure Assessment
Research Division

Enclosure

COMMENTS ON DALLAS LEAD STUDY

Assuming that the questions detailed below can be adequately answered, it is our opinion that the methods used to gather the data were satisfactory. It is also our opinion that because of the design of the study, conclusions concerning geographic distributions of lead are not possible. Also if certain ancillary information could be provided, the efforts to date probably represent an adequate preliminary study.

1. Locations other than Dallas were not evaluated pending receipt of additional information and answers to those questions identified below. Most of the information available for the non-Dallas locations was in summary or abstract form and not of sufficient detail for evaluation.

2. Sampling-in general, there was insufficient detail available to adequately evaluate the sampling methods or design. For example, no sampling plan was provided, therefore sources of variation due to sampling could not be delineated.

a. Map not detailed enough.
b. Actual sample locations not adequately described.
c. Criteria for site selection not given.
d. Representativeness or variability of samples not discussed.
e. Sampling protocols - QA not discussed.
f. Importance of litter or sample depth not discussed.
g. Cleaning procedures for corer and sample container not addressed.
h. Method blanks for the various sampling methods or media were not discussed.
i. Control area - volume and traffic patterns vs experimental area not discussed.
j. Mobile vs stationary lead sources not discussed.

3. Analytical

a. Lead should have been reported as extractable lead.
b. Speciation not discussed.
c. Reference for extraction method not given.

d. QA details not provided -

1. no methods blanks
2. no QA data presented
3. details of what or how extraction factors were used
4. replication of AAS results unclear
5. source of calibration standard not provided
6. independent calibration of standard curves not discussed

e. Need to review raw data and QA data in order to determine quality.

There was $9,000.00 allowed for chemical analysis, and all things considered, the Region got its moneys worth.

4. Statistical Methods

Some of the data were evaluated using Duncan's multiple comparison test. Four areas were compared for various media. All media were highly variable (except water) which made statistical inference difficult. (soils were most variable).

Control N = ~54
2 = ~84
3 = ~84
4 = ~84

The data generally support the conclusion that lead in the experimental areas is greater than the control area.

	1	2	3RSR	4	
Soil	41.62	130.0	493.	829.	μg/g

5. Drinking water

Determine the hydrological and geological characteristics of the area from available sources (e.g. USGS) and determine potential for ground water contamination. Examine available data on ground water usage in the area. Determine extent of soil column contamination (soil profile). Make recommendations from the above assessment.

General Comments

1. Compare blood and soil lead levels with other similar urban areas. Get overall perspective of Dallas Problem

*No air sampling

2. Could assess the feasibility of determining lead source through speciation or isotope ratios.

3. Study probably could be used for the design of further more detailed studies.

4. Chain of custody and documentation not discussed.

Ex5

(109)

QA3

U.S. ENVIRONMENTAL PROTECTION AGENCY
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ENVIRONMENTAL MONITORING SYSTEMS LABORATORY-LAS VEGAS
P.O. BOX 15027, LAS VEGAS, NEVADA 89114-5027 • 702/798-2100 (FTS 545-2100)

Date: MAR 17 1982

Reply to Attn of: QAD

Subject: Support for Dallas Lead Study - Response to Verbal Request from Lamar Miller on March 10, 1982

To: Files

The Laboratory was contacted on March 8 by Matt Bills, Richard Stanley and Courtney Riordin and advised of a potential request to support the subject study. On March 9 we were advised by Matt Bills to support a request from Lamar Miller that would be forthcoming. On March 10 we received a verbal X request from Lamar Miller for technical support to evaluate data collected in the Dallas Lead Study. Three areas of support were requested - 1) QA evaluations of the labs that provided data, 2) evaluation of the sampling strategy, and suitability of the available data for drawing conclusions on environmental contamination by lead in the Dallas area, and 3) preparation of the soil lead results (based on existing data) as isopleths on maps and, if necessary, recommend a soil sampling plan for a more comprehensive assessment. Since the overall objectives were not clearly defined I made several phone calls to clarify the issue. The following is a summary of actions up to March 12.

Called Woody Martin, 382-3119, March 11, 1982

Public pressure was initially created by a newspaper article on lead contamination in the Dallas area. The city of Dallas had collected blood samples that indicated lead contamination. Since some of the blood-lead levels were high, the City had also collected environmental samples to determine if there was an environmental source of lead that was related to these high blood-lead values. The City of Dallas ultimately collected approximately 11,000 blood samples from Dallas residents and had them analyzed for lead. The blood-lead analyses were performed by ESA in Lexington, MA. EPA (Joe Carra, OTS 382-3386) and CDC have received and are reviewing the blood data. Environmental samples (soils, dust, water) were collected and the analyses were performed by the University of Texas at Arlington (UTA) and the City of Dallas. It is not clear if the initial samples were analyzed by both the UTA and City laboratories. Since then, the city has collected additional environmental samples near the residences of children with high blood-lead levels. These data are not available at this time.

The EMSL-LV was asked to review the available data to evaluate their quality and to determine if the sampling strategy was adequate to define environmental contamination. If possible, the data should be mapped in isopleths to identify gradients in the Dallas environment. If necessary, recommendations for additional sampling should be provided to assure that adequate data are available to identify environmental sources of lead. The EMSL-LV was also asked to perform on-site evaluations of the analytical laboratories - City of Dallas, University of Texas, and ESA - that performed the lead analyses. Woody identified Al Davis as the point of contact in Region 6 and Joe Carra as the point of contact in OTS. He did not know if the samples were collected under chain-of-custody and did not indicate that we should look at chain-of-custody during our lab evaluations.

Called Al Davis, 729-2730, March 11, 1982

Al stated his understanding was that CDC was responsible for evaluation of the laboratories with respect to blood data. We were only responsible for evaluation of the laboratories with respect to analysis of environmental samples. His information was that 189 of the individuals sampled had high blood-lead levels. The City of Dallas has collected 100 additional soil samples from areas around the homes of these individuals. The analyses are complete for 50 of these samples and Region 6 was awaiting the results for the remaining 50.

The CDC contacts were identified as Vernon Houck (236-4111) and Dan Vandermeer (236-4113). Al's understanding was that ESA was a certified CDC lab for blood analysis and that we were not to evaluate them. The contacts on Al's staff are Norm Dyer (729-2734) and Larry Thomas. They have forwarded the analytical protocols/QA plans used by the UTA and the City of Dallas for environmental analyses to Paul Richitt and for blood-level analyses to Joe Carra. We will try to review these packages prior to the lab evaluations.

I asked Al to define the purpose of this study as he saw it. His response was: (The State of Texas, County of Dallas, EPA and CDC are reviewing the data to determine if a health problem, due to lead in preschoolers, exists in the Dallas area. If so, could an environmental source of this lead be identified. Our job was to assure that the data being used were reliable and could be used to support policy decisions).

Called Dan Vandermeer, 236-4113, March 11, 1982

Vernon Houck was present during our conversation. Their reveiw indicates that 1806 children under 6 were sampled. Of these, 33 had high blood-lead levels. Soil data, from CDC's perspective, was not high. They questioned the soil data because of 5-fold differences in data from the front to the side of some homes.

CDC did not feel that an on-site evaluation of ESA by EPA was necessary. ESA is a CDC-certified laboratory for blood-lead analysis and is one of their best labs. CDC has performance data for ESA over the past several years. They would be happy to accompany us on the lab evaluations. Vernon advised me that Walt Kovolak (OTS) was organizing a panel of experts to review the data and conclusions of the Dallas Lead Study.

Called Lamar Miller, 382-3062, March 11, 1982

This was a conference call with Paul Richitt to advise Lamar of what had occured. Lamar was advised that CDC felt that there was no need to evaluate ESA. He stated that Marilyn Bracken (OTS), who was responsible for this effort, wanted ESA evaluated. I asked Lamar to confirm this with OTS to avoid any conflict with CDC. He agreed and promised to call the next day to let us know. Lamar did call the next day to advise us that we should evaluate ESA.

I described the purpose of this study, as stated by Al Davis, and Lamar agreed that it was correct. Lamar also indicated that Dr. Hernandez would have to appear before Congress in early April and would like to have our comments in order to be prepared for questions on the Dallas lead problem.

Called Joe Carra, 382-3886, March 12, 1982

Joe has only the blood-lead data. He does not have copies of the analytical protocols or any QA/QC information. Joe was reluctant to provide any of the data because of its sensitive nature. OTS has data from 11,089 participants and Joe did provide this general overview:

1. Four percent of participants had blood-lead levels $\geq$ 30 ug/dl
2. Blood-lead arithmatic mean for all data = 12.7 ug/dl
3. Variance across all data (from mean) = 37.6 ug/dl
4. As an example of distribution of data, approximately three percent of participants fall within a bar distribution of 34-37 ug/dl. Approximately four percent of participants fall within a bar distribution with its midpoint at 5 ug/dl

Joe felt that it was important for us to look at the quality of all of the data as well as evaluate all of the labs who provided data including ESA. He emphasized that we should also review the quality of the analytical data that are still being obtained.

OTS also had data on free erythrocyte protoporphyrin (FEP) levels in each of the blood samples taken. FEP data when combined with the blood-lead data can be used to verify lead toxicity in humans. Joe also suggested that any further studies should include analyses to speciate the various types of lead compounds in the environmental samples. The intent would be to determine if the environmental samples have lead species that are characteristic of an environmental source such as auto emissions, paint or lead smelters.

Joe indicated that OTS has thoroughly reviewed and organized the blood-lead data and has developed maps indicating concentration versus residential location of the individuals sampled. Their results imply a trend of decreasing blood-lead levels away from lead smelters in Dallas. Paul Richitt's staff should coordinate with Joe to assure that we take advantage of the work OTS has already done.

Called Norm Dyer, 729-2734, March 12, 1982

I advised him of our plans for the lab evaluations. He will coordinate with the labs. He has audit information from Dr. Spittler, S&A Director, Region 1 (828-6260), who reviewed ESA's work while it was in progress. Norm will send us what information he has. Ray Lozano, Region 6 QA Officer, will accompany our team on the lab evaluation.

Called Matt Bills, March 12, 1982

This was a conference call with Walt Petrie and Jim Mullins. I summarized our plans and progress. Matt had no problem with our approach. He will ask Lamar to document his request for support, including specific requirements. Matt will check to see if Dr. Mushback (I'm not sure if this is the correct name), an Agency consultant from the University of North Carolina who is an expert on blood-lead analysis, can accompany our team on the evaluation of ESA. Matt will verify that there are no conflicts with CDC related to our evaluation of ESA.

Eugene P. Meier

Eugene P. Meier, Ph.D.
Director
Quality Assurance Division

Enclosure

cc:
Glenn E. Schweitzer, ODC
Richard Stanley, ODC
Paul Richitt, EAD
Walter Petrie, OPM
Steve Simon, LEMSCO
Werner Beckert, QAD
J. Gareth Pearson, QAD

Summary of Status of Dallas Lead Study

I. Purpose. The State (Texas), County (Dallas), EPA and CDC are reviewing the available blood-lead and environmental data to determine if a health problem due to lead exists in the Dallas area. If a health problem exists, they want to identify if there is an environmental source of the lead. Our job is to determine the quality of the data to assure that only reliable data is used for any correlations, conclusions, and Agency policy decisions.

II. Actors.

OTS - Marilyn Bracken, overall responsiblity for the Agency.
Walter Kovalick, Jr.
Joe Carra, 382-3886

REGION 6 - Al Davis, 729-2730, responsible for Region 6 participation.
Norm Dyer, 729-2734, coordination with Region 6 and City of Dallas.
Larry Thomas

CDC - Vernon Houck, 236-4111, responsibility for CDC.
Dan Vandermeer, 236-4113

ENFORCEMENT - Lamar Miller, 382-3062, coordination of Agency activites.
Woody Martin, 382-3119

III. EMSL-LV Objectives.

A. Evaluate the analytical laboratories that performed analyses for the Dallas Lead Study.

1. City of Dallas - environmental samples
2. University of Texas at Arlington - environmental samples
3. ESA, Lexington, MS - blood samples

B. Review the QA/QC information in order to judge quality of the data provided.

C. Review the data and sampling strategy to determine if they adequately define the environmental problem. If possible, map the soil data in isopleths to identify concentration gradients. If necessary, recommend additional soil sampling that may be required to adequately define sources of lead in the environment (Dallas area).

IV. Outputs. Lab evaluation reports for three laboratories - ESA, University of Texas at Arlington, and City of Dallas. Data Review Report which addresses sampling strategy, quality of all of the data, and reliability of the soil data for establishing concentration gradients of environmental lead in the Dallas area. Soil sampling plan, if necessary.

V. Use of Outputs. Our outputs will be used by OTS to define the reliability of data that are used to support Agency policy decisions on lead contamination/health problems in the Dallas area. Our reports and Agency conclusions, recommendations and policy decisions will be reviewed by a panel of experts that is being organized by the OTS.

EX 7C 3 9A4

APR 2 8 1982

EAD

Evaluation of the Sampling Methods and the Monitoring Design Used to Identify Environmental Lead Levels Near the DMC, RSR, GBC, and REF Sites in Dallas, Texas

W. Lamar Miller, Acting Director
Office of Waste Programs Enforcement (WH-527F)

X Enclosed is our critique of the environmental monitoring studies conducted in the vicinity of the four sites in Dallas, Texas. Assuming that the analysis of the samples collected from the two previous sampling studies are accurate, we found that the sample collection techniques used were satisfactory, and that the preparation of sample containers, sample packaging, and sample handling were satisfactory. However, there was a lack of sampling quality assurance for all of the environmental media collected. For example, duplicate or triplicate samples were not collected nor were there any attempts to evaluate and identify decontamination procedures used to clean the soil sampling equipment.

The soil sampling scheme used by the city of Dallas and the University of Texas-Arlington was not adequate to delineate geographical areas of lead contamination nor was sufficient data collected to draw or identify isopleths within any of the study sites. A soil monitoring strategy that will provide the necessary data for identifying geographical areas of contamination and isopleths of soil lead levels in each of the study sites is included in the enclosed evaluation.

The tap water lead levels from the DMC, GBC and RSR study sites were not significantly higher than the tap water lead levels collected from the REF study area. Therefore, until the results of the analytical laboratory evaluations are reported, collection of additional water samples is not recommended.

The dust sampling effort, in itself, cannot be used to fully evaluate the respirable particulate fraction. If monitoring in this area is to be continued we recommend design and initiation of a complementary indoor-outdoor air sampling program.

EAD:[illegible]:2214:cc 4/16/82
EAD:RICHITT
ODC:SCHWEITZER

Sampling quality assurance is a vital part of any scientifically designed sampling and monitoring effort. As such, we strongly recommend that adequate quality assurance be incorporated into any future monitoring studies.

Glenn E. Schweitzer
Director

Enclosure

APPENDIX A

CRITIQUE OF ENVIRONMENTAL LEAD MONITORING STUDIES CONDUCTED IN THE VICINITY OF SECONDARY LEAD SMELTERS IN DALLAS, TEXAS

DALLAS LEAD STUDY

INTRODUCTION

Data collected by the City of Dallas and the University of Texas at Arlington have shown some soils collected in the immediate vicinity of the Dixie Metal Company (DMC) and the RSR Corporation (RSR) have lead levels that exceed 1,000 µg/g. A sampling scheme was designed and implemented by both entities that identified lead levels in a variety of media including tap water, house dust and house paint in the areas surrounding these two industrial facilities. Due to the behavior of airborne lead from industrial facilities such as these, the sampling area was confined to a 2-mile radius around each of the sites. To evaluate the degree of contamination of these two industrially exposed sites, a site identified as the reference area (REF) was selected near the intersections of Lake June and St. Augustine Roads. This reference area was judged by available windrose data to be out of the probable deposition pattern of the industrial sites. The sampling scheme, except for the total number of samples collected in the reference area, was identical to that used at the DMC and RSR sites. In addition, a 2-mile area surrounding a third industrial site, the General Battery Corporation (GBC), was sampled using the same scheme as previously mentioned.

Sampling Scheme

Sampling sites were selected using a stratified random sampling design. The study perimeter included all of the area lying within a 2-mile radius of the suspected contaminant source. From the center of the study area, 16 equally-spaced radii (subtending an angle of 22.5°) were drawn. The study area was further divided by 20 concentric circles spaced from the center at 0.1 mile increments. Each of the resulting sampling sectors were coded by direction and distance from the center. The sectors were selected for sampling by standard random techniques with the following restrictions:

1. At least two-thirds of all sampling sectors were to be in the major downwind direction (Wind Rose Data From Dallas Love Field 1961-1973).
2. Residential and non-residential sites must exist in the sector sections.
3. Sampling sectors were judged eligible for repeat selection only if they were geographically located within 1/2-half mile from the center of the sampling area or if the sector contained both residential and nonresidential sites.
4. Twenty (20) soil sampling locations were selected.
5. Ten (10) of the locations were selected at residences and ten (10) were located at nonresidential sites, such as, schools, parks, playgrounds and/or daycare centers.
    a. Cold and hot tap water was collected from the residential sites.

   - b. Dust was collected from residential sites which included swipes and samples from dust collecting trays.
   - c. Paint chip samples were collected.

The selection of the specific site within the sampling sector was determined by the on-site investigator. Guidelines used included:

1. Permission of home owners
2. Physical biases
   - a. Sites judged too close to major roads were not selected.
   - b. Obvious contaminated areas (oil spills, etc.) were not selected.

SAMPLING METHODS

Soils

A maximum of six and a minimum of four sampling sites were selected at each residential location. Two of the samples were collected in the front yard, two in the backyard and two in the garden, if present, at each residence. At the nonresidential sites the number of sampling locations were selected in part by the activity patterns of children since they were judged to be the critical receptors.

A stainless steel corer with an inside diameter of 3/4-inch was used to collect the soil. The corer, marked in 1-inch increments to a depth of 3 inches was pushed into the soil. After the corer was withdrawn, the soil was removed by a plunger and disected into three 1-inch depth increments. Each increment was treated as a separate sample.

Documentation of sampling locations included site description forms showing the specific sampling site relative to structures, trees and roads. Photographs were also taken of some of the sampling sites.

Guidelines used in sampling site selections included:

1. Samples collected were spaced at least 20 feet from any painted surface.
2. Sampling sites were located as far from areas of vehicle activity as possible, i.e., streets, driveways and parking areas.
3. Sampling sites selected were at least 4 feet from structures in which paint chips might occur.
4. Collection of samples under or immediately adjacent to trees and shrubs were avoided when possible.
5. Sampling sites were selected to represent conditions of the yard, i.e., if yard was barren of growth the site was selected in a barren area.

The techniques used by the City of Dallas and the University of Texas, Arlington, to collect the soil samples are commonly used by soil scientists, as such, the use of the soil corer was judged to be satisfactory. The extraction of soil from the corer, the disection of soils into the 1-inch increments and the subsequent packaging and handling of the soil samples was also judged to be satisfactory.

Poor sampling techniques were judged to be in the following areas:

1. Soil Variation - No attempt was made to obtain a representative sample or to evaluate the wide variation in lead levels that existed in a number of soils collected from the same site. For example, two soil samples collected from a residential lot at 225 Slade Street contained lead levels of 564 and 4,909 μg/g, respectively. Soil lead levels reported from a number of other sites were also of the same magnitude of variability.

2. Cross contamination - The sampling equipment was washed and dried after use at each site. The washing and decontamination of sampling equipment is mandatory for the prevention of sample cross contamination. However, there was no attempt to evaluate or identify whether the washing and/or drying technique selected was adequate. Swipes and samples of rinsings should have been collected and analyzed.

TAP WATER

Both hot and cold tap water was collected in the kitchens of each residence. The tap was opened for a minimum of 1 minute before any of the samples were collected. The samples were collected in 500 ml acid-washed polyethylene bottles. Prior to sample collection the bottles were rinsed three times with either hot or cold tap water. After collection the samples were acidified (1%) within 90 minutes using concentrated reagent grade HNO_3. The techniques used to collect tap water samples, their packaging and handling, are accepted methods for the collection of water samples for metal analysis.

As far as can be determined from the available data, sampling of domestic water occurred only one time. Also, it appears that duplicates or split samples were not collected.

There was no significant difference in the lead levels in cold tap water between any of the study areas (GBC, DMC and RSR) and the REF study area. Also, the data shows that there was no significant difference in lead levels in hot tap water samples collected from the GBC, REF, and DMC study sites. However, the hot tap water lead levels were significantly lower in RSR study site samples than those from the GBC, REF and DMC study sites. As such, collection of additional water samples is not recommended.

DUST AND PAINT

Swipes of household dust were collected from windowsills and tops of refrigerators. Where applicable, swipes were collected from windowsills of inside play areas and children's bedrooms. To supplement swipes, a dust collecting tray was placed upon the refrigerator top for a 28-day period.

Swipes of household dusts were collected by two methods. One involved the use of a commercial towelette (Dioparene) and the other was a Kimwipe moistened with distilled water. After collection, the moistened collectors were placed in sample containers until analyzed. The dust collection tray was an acid-washed rectangular polyethylene container. After the 28-day collection period the tray was sealed with an acid-washed polyethylene lid until analyzed.

Paint chip samples were collected from residential areas which showed flaking on the walls or windowsills. A small pocket knife was used to collect approximately 1 gram of paint chips. The paint chips were placed in plastic containers until analyzed.

As per citizen's request, outdoor dust swipes were collected. The collection of outdoor dusts were primarily confined to automobiles. However, one 8-foot canopy was also swiped.

The location of the swipe samples was recorded. Also, the dimensions, approximately 100 cm^2 of the area swiped, was measured and recorded.

Swipes and dust collecting devices are commonly used and may provide needed information concerning exposures of particulate contaminants by the inhalation pathway. However, in an active household in which significant volumes of indoor and outdoor air are mixed, the heavier particulates would tend to settle out first, thereby possibly biasing the data obtained from this sampling technique. Using only this method, adequate assessment cannot be made for the smaller and more respirable particulate fraction. For example, studies reported by Boggess 1977 have shown that particles with diameters of less than 10 μm are not significantly affected by gravitational settling. Also, particulates less than 10 μm are generally considered to be the most respirable.

Analysis of the dust collecting media, Dioparene towelettes and Kimwipes, should be completed to identify and evaluate their lead content and their suitability as dust sample collectors.

If warranted, a monitoring scheme should be designed that will address outdoor and indoor air sampling complemented by dust sampling. In addition to total lead analyses of these samples, analysis should include size determination and chemical characterization.

SOIL SAMPLING DESIGN

The Environmental Monitoring Systems Laboratory in Las Vegas was requested to review the collected soil data, draw lines of constant concentration of lead (isopleths), critique the monitoring scheme used, and recommend additional soil monitoring if necessary.

Data collected from previous soil sampling studies have indicated that higher lead concentrations were generally found in those soils collected in close proximity to the suspected source. As distance increased from the suspected source the soil lead levels generally decreased. Also, the data indicates that a depth migration of lead by some means, probably mechanical, has occurred. However, the sampling scheme used is not adequate to delineate geographical areas of lead contamination, nor was there sufficient data collected to adequately draw or identify isopleths. Therefore, even though requested, no isopleths were drawn.

The data was analyzed by a one-way analysis of variance. These analyses, as shown in Appendix A, show that the reference area (REF) mean lead level is statistically significantly lower at the 95% confidence level than the GBC mean lead level. Also, the GBC mean lead level is significantly lower at the 95% confidence level than the mean lead levels of both the DMC and RSR sites. In addition, the data shows that there is no statistically significant difference at the 95% confidence level existing between the mean lead levels of the DMC and RSR sites. However, 1,000 µg/g of lead is above the 99% confidence interval of the mean of either the DMC or RSR sites.

In summary, these data show that the DMC and the RSR sites belong to a different and higher population of lead levels than the GBC or the REF sites. All sample means were less than 1,000 µg/g and statistically significantly below 1,000 µg/g. Thus, since GBC is significantly lower than either DMC or RSR additional sampling may be justified in the RSR and DMC sites but no additional sampling is justified in the GBC site.

Maps of the four study areas were made. Soil lead levels reported by the City of Dallas and the University of Texas at Arlington were plotted showing the approximate collection locations and the mean values calculated from the surface soils collected at each location. The mean levels were then ranked as a function of distance from the suspected source. The information obtained from this ranking was used to construct a semi-variogram for each of the study areas as shown in Appendix B. Using this data and the statistical methodology of kriging, sampling grid dimensions were calculated. Statistically, kriging is designed to make use of the dependence of one sample with other closely associated samples and is able to produce unbiased estimates from the data. It not only will estimate the values of spatially distributed variables but will also identify the probable error associated with the estimates.

The calculated and recommended sampling scheme to identify the geographical extent of the soil lead contamination is as follows:

1. The dimensions of the square grid should be 750' x 750'. The total area covered by the grid will be contingent upon the minimum lead concentrations of concern. The grid should also be oriented if possible along the axis of the plume.

Based on the plotted data for the DMC study area, it appears that soil levels similar to REF levels would be found if the grid extended north of Ervay and Beaumont Streets, and south of Southerland and Bonnie View Road intersections. The western boundary should extend to Highway 342 and east of Highway 45. Examination and identification of lead from nearby major vehicle arteries may require an eastward extension of the grid to Highway 75. The outer limits of the grid can be extended or decreased as warranted. Extension of the grid to the west may be necessary due to the high lead levels 731 and 1,249 µg/g found at DMV-11. These two values should be verified by reanalysis of extract if available or by additional samples before extending the grid.

The soil lead levels appear to be more localized in the RSR study area. As such, soil levels similar to REF levels would probably be encountered if the grid extended 1.0 miles to the east, west, and southern directions and 1.5 miles in the northern direction from the RSR facility. Extension of the grid can be increased or decreased as warranted.

It is recommended, as previously suggested, that sampling be terminated at the GBC site as the soil data doesn't indicate excessive lead levels in this area.

It is recommended that the REF grid dimensions (750' x 750') be identical to that used for the DMC and RSR sites. The geographical location of the REF site appears to be satisfactory.

2. Each of the grid intersections should be identified by map coordinates for future data interpretation. Soils within a radius of 30 meters of each of the grid intersections should be sampled using procedures identical to those described by the City of Dallas. The 30-meter radius from the grid intersections will provide on-site flexibility for selection of suitable sampling sites. The use of the core sampler and the sample handling and chain-of-custody technique used should be adhered to. Data received by EMSL-LV does not show or indicate any problems were encountered in these areas with soil-organic layers. As such, only mineral soil should be collected. Also, only the upper 3 inches should be collected (see Appendix C). If organic or vegetative layers are commonly

encountered a sampling scheme would have to be designed to address this parameter. The on-site investigator shall be responsible for determining the specific sampling site. Criteria used for making this determination will be identical to those previously used and identified in the University of Texas sampling protocol. Documentation at each site shall include photographs showing sampling area, a schematic showing sampling locations, including dimensions and other pertinent information including structures, canopy layers, ground surface characteristics, streets, driveways, etc.

Following the selection of a specific sampling area, four subsamples should be collected. As noted in Appendix D, calculation of the in-yard variance of the surface soil samples was less than the between-yard variance suggesting that four subsamples per sampling site is adequate. The subsamples should be collected 15 meters from the center of the sampling area, one each from the north, south, east and west directions. The corresponding depth increments should be placed in appropriate sample containers and mixed prior to analysis. To obtain a homogeneous sample, it is recommended that mixing be completed in the laboratory.

We recommend that sample integrity be maintained as per EPA NEIC chain-of-custody requirements, and that recommended sample containers, preservation and holding time requirements be adhered to as per the Federal Register, Vol. 44, No. 233, 1979.

PRECISION AND ACCURACY

It is recommended that precision and accuracy of the sampling procedures be addressed. The degree and level of precision and accuracy is dependent on the ultimate use of the data. Recommended additional samplings for addressing precision and accuracy include:

1. Collection of duplicate samples.
2. Collection and analysis of splits.
3. Analysis of spikes and field blanks.
4. Decontamination samples, such as equipment swipes, rinse water and/or solvents used in equipment cleaning.

Literature Cited

1. Boggess, R. W. Editor. Lead in the Environment. 1977. Chapter 7. Transport and Distribution from Mining, Milling and Smelting Operations in a Forest Ecosystem, p. 139. Prepared for the National Science Foundation Research Applications Directorate.

APPENDIX A

Pollution observations are often from a logarithmatic normal rather than a normal distribution. To test which distribution is Dallas' lead, the skewness was calculated. The skewness was improved by the log transformation and the transformed data was used in the analysis of variance and other statistical tests.

The original sampling had two aberrant observations in DMC at 14-HS. These were resampled and the new observations replaced the original values.

The analysis of variance showed statistical significance (at 95%, 98%, 99% levels) between areas.

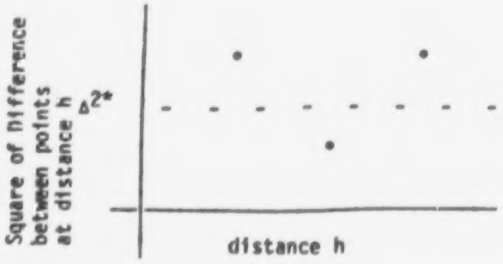
Duncan's multiple range test shows three populations as diagrammed below:

$$\underline{\text{REF}}\quad\underline{\text{GBC}}\quad\underline{\text{DMC RSR}}$$

At the 95 percent level DMC and RSR are from one population of lead observations shown by one underscore, but GBC and REF are different populations discrete from each other and each represented with its own underscore.

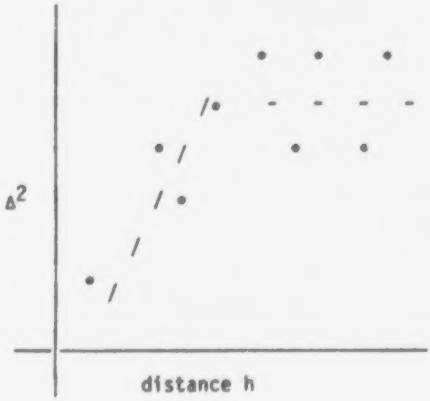
APPENDIX B

How far apart should the samples be? Classical statistics deals with random variables and computes how many samples should be taken. Geostatistics deals with regional (or spatial) variables and computes how far apart samples should be taken. A regional variable is autocorrelated in time or space or both. Thus, a lead observation is a regional variable if a sample value is numerically closer to spatially nearby samples and is numerically different from remote samples. Geostatistics examines this correlation structure by means of a semi-variogram. This is a graph of the square of the difference between samples at distance h apart plotted against distance h. If the samples are uncorrelated the variogram is a horizontal line. Interestingly, the variogram of REF was a horizontal line. This indicates that the lead



*(square of difference in concentration between pairs of all points at distance h)

values found in REF were not structured. This is what should be found in a control area. If the samples are correlated, the first values of h-distance have rising or increasing squares of differences. At greater h-distances their squares of difference become horizontal as in the previous variogram. The first or rising part of the plot is the informative part because it measures the range of correlation. This is the distance before the plot becomes horizontal. The distance between sampling points must be about 2/3 of the range of correlation. Interestingly, both DCM and RSR had this type of semi-variogram (called spherical or Metron model). This shows there is structure or autocorrelation to the lead samples in these sites. The ranges of correlation at the two sites were about 1,200 feet and thus the recommended grid distance between sampling points is 750 feet.



APPENDIX C

How deep a plug should be taken for a sample? It is most important that all samples must be taken with pluggers of identical inside diameter and depth. Obviously all samples will be compared for national background levels or world averages. This requires uniformity. However, since nine profiles with lead content reported inch by inch of depth were done in Dallas, this opportunity was taken to evaluate the use of 3-inch plugs.

Studying the data, the first three depths (1"-3") are representative of higher values. In general, lower depths (4"-6") drop off in lead content. Since lead is usually in insoluble forms this is expected. DMC-14-HS-04 and DMC-17-HS-01 are exceptions to this generalization because their highest values are at inch four; however, even in these exceptions, inch five belongs to the lower range of lead content. Cultivation or other disturbances would physically explain these anomalies. A one-way analysis of variance shows the lead values of the top 3 inches to be statistically significantly higher at 99 percent confidence level than the lead values of the bottom 3 inches.

APPENDIX D

How many samples should be composited to describe a sampling site? This is a very important question and should be answered in terms of spatial variables (see Appendix B). Because of the time limit, this question was answered in terms of random variables. Within an area (e.g. DMC, RSR) the between-site-variance was statistically significantly larger at the 95 percent level than the within-site-variance. A two-way analysis of variance tested this. This implies that additional effort should be invested initially in sampling more sites in order to reduce the between site variance since it is the larger. This implies that the within-site-variance is adequately described by four (4) samples per site.

654

CHEMICAL WORKPLAN

FOR LEAD

REGION 6

MAY 1980

CHEMICAL WORK PLAN

OUTLINE

1. Executive Summary

II. Introduction

III. Sources of Environmental Lead

IV. Sources of Exposure

V. Health Effects

VI. Population at Risk

VII. Ecological Effects

VIII. Environmental Contamination

IX. Principal IRLG Actions & Current Status

X. Current EPA Controls

XI. Source Contributions to Daily Lead Uptake in Humans(23)

XII. Conclusions and Deficiencies

XIII. Rationale and Implementation Plan

XIV. Lead Manufacturing, Production, Usage and Emission Sites (NPDES permits noted Appendix A)

XV. Areas for Location of Point Source Monitors In Texas (Appendix B)

EXECUTIVE SUMMARY OF CHEMICAL WORKPLAN
FOR
LEAD AND ITS COMPOUNDS

Statement of Concern

The potential for lead poisoning in sensitive populations is a major concern, particularly in children, pregnant mothers, and occupationally exposed workers in urban environments where elevated levels of lead in air, dustfall, and soil represent significant additional sources of lead intake.

In the Preamble to the National Ambient Air Quality Standard for lead the Agency recognized the potential problems in the attainment of the standard in the vicinity of certain facilities and included flexibility for action by Regional Administrators and States.

In addition, none of the National Media Programs address soil as a source of exposure for sensitive sub-groups of the population.

Health and Ecological Effects, Environmental Behavior

Clinical effects of lead poisoning may include acute and chronic central nervous system damage, peripheral nerve paralysis, kidney damage, and damage to blood formation processes which may lead to anemia. The risk of clear-cut clinical effects is greatest when blood lead levels increase above 80 ug/100g whole blood, and at lower blood levels when anemia prevails. Mild poisoning may be reversible if exposure to lead is discontinued. A single episode of lead poisoning in a child may result in brain damage ranging from subtle learning disabilities to extensive mental handicaps. Maternal over-exposure may have greater toxic effects on the fetus than on the mother.

Common sources of livestock lead poisoning include lead-based paints, storage batteries, used motor oil and airborne lead contamination of hay and pasture vegetation from smelting and other lead industries. A daily lead intake of 6-7 mg/kg body weight has been suggested as the minimum level which may result in poisoning in cattle.

Sources, Environmental Levels

U.S. consumption of lead was 1.6 million short tons in 1974. Major uses included storage batteries (53 percent), gasoline additives (16 percent), pigments (7 percent), ammunitions (5 percent), solder (4 percent), and cable covering (3 percent). The elevated atmospheric levels of lead in larger cities was attributed primarily to motor vehicle combustion of leaded fuel additives. About 180,000 tons of lead are emitted to air annually from motor vehicle combustion of fuel additives. Other sources of lead pollution include coal and fuel oil combustion, lead smelting, incineration of solid wastes, and lead paints.

Lead levels in ambient air averaged 1-3 ug/m^3 in urban areas, 0.1-0.5 ug/m^3 in surburan areas, and less than 0.05 ug/m^3 in rural areas, except in the vicinity of point sources and heavy traffic. The levels of lead in natural lead ore deposits and point source discharges.

The average daily oral intake of lead is estimated at 300 ug for adults, and 130 ug for children. Childhood and infant pica has resulted in numerous cases of lead poisoning from the ingestion of peeling lead paint and other lead containing substances, and the inhalation of street dust.

Population at Risk

Environmental quality standards should be designed to protect susceptible subgroups of the population fully. In the case of Pb, children are both most susceptible and are at a higher risk. Barltrop, studied children and their mothers living in two different towns with differing soil Pb concentrations. The study revealed that the blood Pb levels of the children were higher than that of their mothers in both cities. Landrigan, found a strong inverse relationship between blood Pb levels and age among people living in the same area near a smelter in El Paso, Texas. High blood Pb levels are a health risk to the nervous system. The major development of a child's nervous system takes place during his first four years. This age group represents the highest risk group. Thus, it is imperative that any ambient lead standard be designed to protect young children.

Existing Regulatory Approaches

The Food and Drug Administration has restricted the use of lead in paints to less than 0.5 percent by weight for use on residential surfaces assessible to children, has proposed a tolerance level (0.3 ppm) for lead in evaporated milk and an action level (7 ug/ml) for leachable lead in pottery and enamelware, and is considering limitations below 0.5 ppm for lead in other canned foods. OSHA has proposed an action level of 50 ug/m^3 and a permissible exposure limit of 100 ug/m^3 during an average eight (8) hour work period for lead and its compounds. The Consumer Products Safety Commission is currently undertaking a review of the "safe" level of lead in paints.

EPA has established effluent limitation guidelines on lead discharges from several industrial point source categories. Fifteen lead compounds have been designated as hazardous substances. Special care is required in ocean dumping or other disposal of lead and its compounds. Interim Primary Drinking Water Standards set a maximum contaminant level for lead of 0.05 mg/l.

Pursuant to Section III of the Clean Air Act, EPA has established standards of performance for primary and secondary lead smelters. Lead has been added to the list of air pollutants under Section 108(a) of the Clean Air Act, and a National Ambient Air Quality Standard for lead of 1.5 ug/m^3 has been issued.

Final regulations promulgated on December 6, 1973, control the amount of lead additives in gasoline. Lead levels in gasoline must meet the 1.4 g/gal limitation beginning October 1, 1976.

Current Controls

Lead emissions can be controlled through limitations in both air and water permits. Currently only fourteen (14) facilities (out of about 95 facilities) which have been identified as lead handling or emitting facilities have an NPDES permit which contains a lead discharge limit.

Air emissions which are controlled by the States through the SIPs for lead include only fourteen (14) facilities, which meet the 5 tons/yr criteria (41 other sites are referenced by the States as having significant lead emissions).

Recommendations

1. Evaluate soil/dust lead levels in target areas and initiate appropriate action.
2. Establish monitoring sites in neighborhoods containing lead industries that are not currently included in the SIP's. (Cities with less than a 500,000 population).
3. Installation of monitors in areas around industrial sources of lead emissions not presently covered by SIP's (less than 5 tons/yr-25 tons/yr).
4. Change monitoring sites identified in SIP's to reflect exposure of population at greatest risk by locating monitors near public schools where feasible.
5. Establish State Water Quality Standards for lead in all Region 6 States, except Oklahoma. (Oklahoma already has a WQS).
6. Determine lead contribution from lead pipes to the exposure of sensitive sub-groups.
7. Assess lead contribution from Municipal Incinerators to localized exposures.
8. Evaluate 208 Water Quality Management Plans and incorporate identification of lead sources and an assessment of controls.

Introduction

Lead is unique among the toxic heavy metals in that it is relatively abundant in the earth's crust. World production exceeds 3.5 million tons/year, a far larger quantity than the production of any other toxic heavy metal.

The total environmental exposure must be viewed in terms of natural lead plus lead contamination. The natural concentration of atmospheric lead has been estimated at 0.0005 µg/m as compared to the average of 0.79 µg/m^3 in urban air. Higher concentrations have been reported in heavy automative traffic, 38.0 µg/m^3. The daily inhalation intake at 0.5 µg/m^3 concentration is about 11.4 ug.(1)

The natural lead in fresh water has been estimated at about 1 to 10 ug/liter. In the United States the lead in rain averages 34 ug/l; figures as high as 300 µg/liter have been recorded. However, rain tends to reduce the local air lead concentrations. Fortunately, much of the lead entering the drinking water supply is removed naturally by sedimentation.(1)

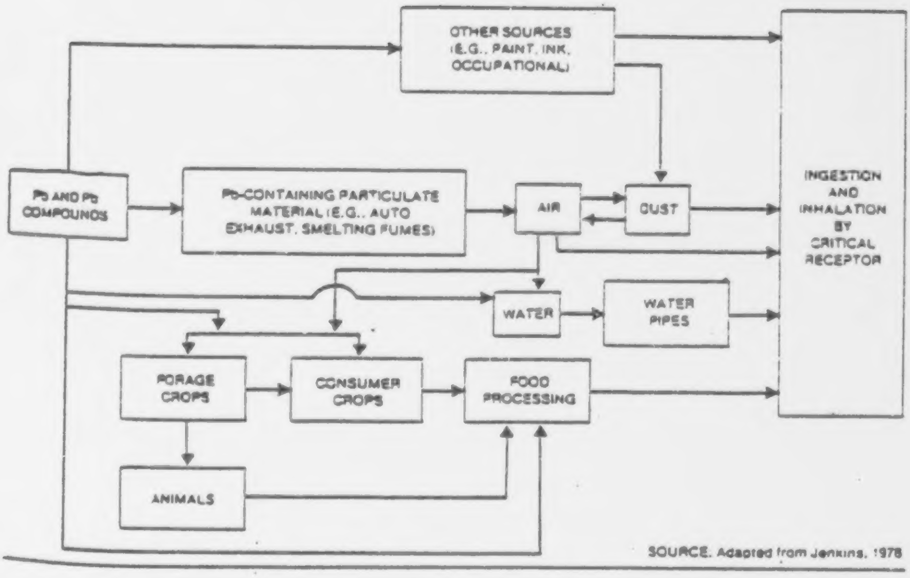
The natural concentration of lead in soils, exclusive of areas near lead deposits, has a range of 2 to 200 ppm, with an approximate average of 16 ppm. The soil lead concentrations decrease dramatically with the distance from roads and the depth of soil.

Plants can translocate soluble lead through their roots to leaves and fruits. Fortunately, there is little translocation of airborne lead from the leaves to the roots. The majority of lead pollution is not available for incorporation into plants because of its lack of solubility.

The body burden is a function of exposure. For example, the American total body burden is 121 mg of lead with an organ concentration of 140 ppm in the spleen. (1)

Sources of Environmental Lead

Humans are exposed to lead and lead-containing compounds through the various environmental pathways. Lead is present in food, water, air, soil, dustfall, paint and other materials which the general population comes in contact. Each of these represent a potential pathway for human lead exposure via inhalation or ingestion as exemplified in the chart below (2)


OTHER SOURCES (E.G., PAINT, INK, OCCUPATIONAL)
Pb AND Pb COMPOUNDS
Pb-CONTAINING PARTICULATE MATERIAL (E.G., AUTO EXHAUST, SMELTING FUMES)
AIR
DUST
INGESTION AND INHALATION BY CRITICAL RECEPTOR
WATER
WATER PIPES
FORAGE CROPS
CONSUMER CROPS
FOOD PROCESSING
ANIMALS

SOURCE: Adapted from Jenkins, 1978

Major Environmental Lead Exposure Pathways

Lead in Ambient Air

The lead used in gasoline antiknock additives represents a major fraction of the total U.S. lead consumption, and motot vehicle emissions constitute the major source of lead emissions to the atmosphere. In 1975 the most recent year for which data are available, approximately 88.8% of total atmospheric lead arose from that emission source(3). The next largest contributors primary copper and lead smelting facilities provided 2.5 and 1.7 percent, respectively of the total (3). Local atmospheric lead pollution from industrial plants processing lead and its products can be severe, but their overall contribution to the pollution load across large areas is minimal(4).

Since the early 1960's the National Air Sampling Network (NASN) has routinely collected and analyzed airborne particulate samples for selected metals.

However, recent studies have indicated that the atmospheric lead constituent of the total suspended particulate (TSP) is comprised of particles whose mass median diameter is approximately 0.2 to 1.43 μ. Up to 74 percent of the particles are less than 1 u in diameter(5). It is widely believed that particles in this size range are easily respirable, and reach the innermost parts of the lungs, since pulmonary deposition of inhaled particulate is greatest in the 0.01 to 2μ range(5). Virtually all lead deposited in the lung is eventually absorbed into the blood (7,8).

In addition to inorganic lead particulate material, the atmosphere may contain organic lead vapors (e.g. lead alkyls) which are not detected by the NASN sampling protocols. Most of these organic lead compounds arise from the production, handling, and use of gasoline containing lead antiknock additives. They are photoreactive and their presence in local atmospheres is transitory. Studies have indicated that lead alkyls represent less than 10 percent of the total lead loading in the atmosphere(7).

According to NASN data, the levels of lead compounds in the atmosphere are slowly decreasing, mainly as a result of the decreased use of leaded gasoline (9). As might be expected, urban atmospheres contain higher lead concentrations than nonurban atmospheres(10). In 1974, the arithmetic mean urban lead concentration was 0.89 $\mu g/m^3$, as compared to the nonurban mean of 0.11 $\mu g/m^3$.

Although the NASN annual averages are useful in identifying trends in air-lead levels, data from individual sampling sites or regional areas must be examined to identify those locations that experience higher than average lead concentration Higher ambient lead levels are usually associated with primary lead smelting operations located in the area.(10)

The particle size distribution of lead aerosols are dependent upon the emission source. Lead particles from vehicular traffic are usually less than 1μ in diameter whereas lead containing particles near primary smelters are in the 7.0μ size range (2). In general these large particles are not likely to penetrate into the pulmonary regions of lungs, they are usually deposited in the nasophanyngeal region and are eventually swallowed or expectorated. These large particles are also more apt to settle out of the atmosphere quickly and therefore contribute significantly to the lead levels in dust, soil, water, and vegetation in the vicinity of the source(11).

In areas close to main transportation arteries, atmospheric lead concentrations have been shown to vary directly with the volume of vehicular traffic and the size of the community(12,7). Mean atmospheric lead levels in such areas have been shown to vary from 9.4 to 38.0 $\mu g/m^3$ according to proximity to freeways, time of day, day of the week, and sampling elevation (12,7)

The NASN sampling sites usually stationed on rooftops, do not accurately depict the situatian at street level.

Other factors impaction individuals exposed to street level vehicular lead particles include the resuspension of particulate lead estimated to be 1 to 5% deposited on and near the road surface, and the effect of airborne street level lead containment within the indoor environment. Again NASN samplers would be insensitive to these factors due to rooftop locations(13).

Lead Concentrations in the Diet

The exact quantity of an adult's total daily lead intake is a function of the type and size of the diet. The occurrence of lead in various foodstuffs may be a result of natural bioaccumulation, deposition of airborne lead particles, and/or food processing and serving.

Since most foods have been found to contain about 0.5 ppm of lead or less, intake depends more upon the size and nature of the diet than on a choice of particular food (14). However, some segments of the population with special dietary requirements, such as infants may consume selected foods found high in lead content (e.g., canned milk). As a result, they ingest more lead than might be expected based on average adult dietary constituents.

Lead in Drinking Water

The amount of lead dissloved in water depends upon the equilibrium constants of the chemical form(s) of lead present. These equilibrium constants depend to some extent, upon the chemical characteristics of the water system. Studies of lead equilibruim solubilities of a variety of inorganic lead compounds show that water could contain from several micrograms to several hundred micrograms of lead per liter of water, depending upon the pH, temperature, and mineral content of the water (15,16).

In a survey encompassing over 700 surface water sites in the United States and Puerto Rico, less than 0.5 percent of the samples exceeded 50 μg lead per liter of water (17).

Lead in drinking water above that concentration found in the make-up water usually occurs as a result of the physical characteristics of the water distribution system. The use of lead pipes within the system provides an opportunity for the lead in contact with the water to go into solution.

In a 1970 study (18) lead concentrations in finished drinking water collected at 969 public water supply systems in the United STates ranged from an undetectable amount to 640 $\mu g/l$. Of the suply systems sapmled, thirty-seven sites (1.4% of the total) contained lead in concentrations exceeding the current national interim primary drinking water standard of 50 $\mu g/l$(18).

Additional Sources of Childhood Lead Exposure

Lead-containing nonfood substances are deliberately or inadvertently ingested by some individuals. This is especially prevalent and frequent in young children (19,20,21), many of whom display patterns of repetitive hand-to-mouth activity, mouthing behavior, or pica. Hand-to-mouth activity coupled with the presence of foreign substances (particularly soil and dust) on the hands and ingestion of soil-and dust- contaminated food may both result in the ingestion of appreciable quantities of lead. Mouthing of substances containing or contaminated with lead, or pica for such substances, may also be a mechanism of lead exposure. The list of substances commonly mouthed or ingested by children displaying these behaviors has been shown to include paper (newspaper, wallpaper, toilet tissue, facial tissue, cardboard), dirt (soil, dust, clay, sand, ashes), paint and plaster, tobacco, and toiletries and cosmetics (22). Some of these are known to contain lead.

Soil/Dust

Human activities are responsible for the major portion of lead found in soil and dust. Natural occurrence of lead in the earth's crust accounts for an average of 15µg/g (16). Mean rural soil and dust lead levels of 60 and 275µg/g have been reported (23). However, lead levels in soil and dust in metropolitan areas are said to be as high as 400,000µg/g(23,43). The mean interior house dust-lead level for an urban area was reported as 11,000µg/g(23). Metropolitan soil-lead levels as high as 3,357µg/g have been reported(6).

The combustion of leaded gasoline is a major contributor to the high lead levels in urban soil and dust (6). Soil lead concentrations diminish with distance from roadways, but increase significantly near dwellings (23), reportedly due to runoff.

Industrial point sources (e.g. smelters and battery plants) are a major local source of soil/dust-lead contamination. Air and soil levels were shown to decrease with increased distance from a smelter site in El Paso, Texas (24).

Other stationary sources of lead emissions include combustion of Waste oil, incineration of solid waste, iron and steel production plants with poor pollution controls, battery manufacturing and lead alkyl manufacturing. Lead that may be emitted to the environment from these operations includes that in stack emissions and fugitive dusts.

Paint

Ingestion of paint by children suffering from pica has been associated with numerous cases of childhood lead poisoning. Epidemiological evidence indicates an association between elevated blood-lead levels and children with pica for paint(6)

Pica for paint is of major concern. Extremely high lead concentrations are found in some older paint coatings and are still available as peeling and flaking paint (indoor and outdoor) on older dwellings. A survey of over 2000 dwellings in Pittsburgh revealed that at least 20 percent of the residences built after 1960 had at least one surface with greater than 1.5 $\mu g/cm^2$ lead (25). Smaller surveys in El Paso, Texas, and Silver Valley, Idaho, found indoor paint-lead concentrations of 1 percent by weight (10,000 $\mu g/g$) (24).

Market surveys in 1971 showed eight of seventy-six paints tested contained 2.6 to 10.8 percent lead. The Consumer Product Safety Commission found that only 2 percent of interior paints tested had lead levels exceeding 1 percent (NAS, 1976). The same survey found that 70.8 percent of oil-based paints and 96.1 percent of water-based paints contained less than 0.06 percent lead (the current paint standard).

Regulation of the use of lead in house paints did not exist until 1955, when a 1 percent voluntary standard was adopted. A federal standard of 1 percent was imposed in the early 1970's lowared to 0.5 percent in 1976, and further lowered to 0.06 percent (600 ug/g) in 1977. It is apparent that paint with lead levels in excess of >1 percent (by weight) is still readily accessible to children who live in older dwellings (26).

Newsprint

Newsprint can contain up to 1 percent lead (by weight). Colored newsprint contains ~~the greatest amounts~~ of lead. Handling newsprint may result in appreciable dermal exposure; and, coupled with immature dietary habits (e.g., licking and sucking fingers), newsprint may represent a source of ingested lead as well. Ingestion of newspaper is common among children with pica. Indirect newsprint-related exposure can result from elevated air lead levels in homes where newspapers and magazines are used as fireplace fuel.

Health Effects

Lead may cause both acute and chronic effects which usually result from the accumulation of lead in the body over a certain period of time. The major effects are related to four organ systems, i.e. hemopoietic, nervous, gastrointestinal and renal.

Chronic effects

Hemopoietic system (28)

Anemia is a common sign among occupationally exposed workers, although it is often so mild or moderate that it may not be detected unless observation on a group basis is carried out. Anemia due to lead is micro-or normocytic (normal red blood cell), but not macrocytic, (abnormally large erythrocyte), being caused by a combined effect of the inhibition of hemoglobin synthesis and shortened life span of circulating erythrocytes (red blood cell). According to numerous studies performed in recent years, many stages in the pathway of heme synthesis are inhibited by lead.

Nervous system (28)

Increased lead absorption may give rise to effects on both the central and peripheral nervous systems. Effects on the centralnervous system which are manifested as encephalopathy (degeneration of brain) are seen more frequently in childhood lead poisoning from pica (eating dirt) or other causes, possibly because children are more sensitive to the action of lead than adults. Subclinical effects on the peripheral nervous system have been reported in occupationally exposed workers.

The so-called chronic encephalopathy may be either an effect on the brain of a long-term exposure to lead or a residual effect of an acute episode of encephalopathy. The severity of encephalopathy depends on a combination of factors which include intensity and duration of exposure, and age.

Milder symptoms of the effects on the central nervous system may include mental deterioration, hyperkinetic or aggressive behavior, loss of appetite, difficulty in sleeping, abdominal pain, and vomiting. However, the etiology (the study of the cause of disease) of such manifestations is not always clear, because those children who suffer from cerebral injury due to traumatic, toxic, viral or bacterial infections also exhibit similiar symptoms.

The Gastrointestinal system (28)

Colic (abdominal pain) is usually a consistent early symptom of lead poisoning, warning of much more serious effects that are likely to occur with continued and prolonged lead exposure. Although most commonly seen in industrial exposure cases, colic is also a lead poisoning symptom present in infants and young children.

Beritic reported on the cases of 13 of 64 men exposed on their jobs to occupational levels of lead. The 13 had colic, probably lead related, and constipation. They had blood lead levels ranging from a little less than 40 to 80 micrograms/dl as determined by polarography, a technique which tends to yield values lower than the actual blood levels. The diagnosis of lead-caused colic was supported by findings of high urinary coproporphyrin, excessive basophilic stippling, reticulocytosis, and some degree of anemia, all of which are other clinical signs of lead poisoning. (28, 29).

The Immunologic System

Recent reports suggest that exposure to lead may interfere with normal susceptibility to infection. Hemphill found that mice injected with subclinical doses of lead nitrate for 30 days showed greater susceptibility to challenge with Salmonella typhimurium than controls that received a saline injection containing no lead. Selye found that rats injected with lead acetate (minimal effective dose of 1 mg/100g body weight)were susceptible to a variety of bacterial endotoxins (toxins produced by the bacteria themselves) to which this species is ordinarily resistant. (29)

Kidney

Long term exposure to lead may give rise to the development of irreversible functional and morphological renal changes. These consist of intense interstitial fibrosis, tubular atrophy and dilatation (28).

There is convincing evidence in the literature that prolonged lead exposure in humans can result in chronic lead nephropathy (disease of kidney). In spite of normal clinical findings, some with very long exposures were reported to have interstitial and peritubular fibrosis, determined by renal biopsy. This pathological finding is commonly referred to as chronic lead nephropathy, which is characterized by slow development of contracted kidneys with pronounced arteriosclerotic changes, fibrosis, glomerular atrophy, and hyaline degeneration of these vessels. This is a progressive disease sometimes resulting in renal failure. It seems to primarily occur in industrially exposed workers and in older adults who have been diagnosed as having lead poisoning early in life. There is also evidence that lead poisoning in children is manifested by the same symptoms (28).

Primary route of exposure for the general population is food, for occupational groups, inhalation; important sources of exposure for children in some countries are lead paint, and soil and dust. Estimated daily intake via food is for adults about 200-300 micrograms; air pollution may contribute up to about 20 micrograms and sources is up to 30 to 40 micrograms (29).

The rate of deposition, retention and absorption of inhaled lead is highly variable, depending on particle size, physicochemical form of lead and the efficiency of lung clearance mechanisms. There is no evidence of accumulation in the lungs and all lead retained is eventually absorbed or transferred to the gastrointestinal tract. For practical purposes it is assumed that, on average, about 30% of inhaled lead is absorbed (28).

About 10% of ingested lead is absorbed in the gastrointestinal tract. This fraction may be higher for infants and children (28).

Lead body burden consists essentially of two compartments: bone (containing about 90% of the total content) with a half-time of about 20 years; the amount of lead in this compartment increases throughout life; and a smaller compartment with a half-time of about 20 days (blood, soft tissue and rapidly exchangeable bone fraction). (28)

Absorbed lead is excreted mainly via urine (about 80%) and gastrointestinal secretion; small amounts are secreted in milk, sweat, hair and nails. Placental transfer of lead has been demonstrated. (29)

Lead poisoning in children is a uniquely neglected public health problem of the 20th century. In an era which boasted of many major advances in preventive medicine, this man-made illness, which is associated with formidable mortality and morbidity, remained for several decades a disease ignored by most health workers. It is only in the last 10 years or so that the problem has begun to receive some long overdue attention. Even so, few are aware that since mass screening began in the United States in 1971, 3,000 to 4,000 children have been treated for lead poisoning each year, while thousands more who have shown evidence of undue lead absorption have received no treatment at all. (30)

Lead poisoning has been known to be preventable since the early 1900's, long before prevention of polio by vaccination became feasible. One may ask: How could a preventable disease which can leave survivors with mental retardation, blindness, cerebral palsy, learning disabilities, behavior disorders and other problems escape national attention for so long? (30)

Recent investigations have also helped explain children's special vulnerability to lead toxicity. It had been known that adults absorb approximately 10 percent of the lead ingested but only in 1971 were metabolic studies conducted which revealed that children absorb approximately 50 percent of the lead ingested and that they retain a much greater percentage of the lead absorbed and store a larger percentage of the body lead burden in their soft tissues, where it is metabolically active. Children are also more sensitive than adults to the toxic effects of lead. (30)

Studies in lower animals further indicate that the brain in the young is much more vulnerable to lead toxicity, findings which support the clinical observation that lead encephalopathy is much more common among children. (30)

A very important recent development has been the focus on the problem of undue lead absorption in young children. The blood lead or EP levels in these children indicate that they have absorbed an excessive amount of lead, although most of them present no overt clinical evidence of lead poisoning (30).

Interest in this area intensified following discovery, in the late 1960's that this phenonomenon is markedly prevalent and geographically widespread. Expanded screening programs revealed that a majority of children found to have evidence of undue lead absorption did not meet current diagnostic criteria for lead poisoning and so received no treatment. For the thousands of children uncovered by screening so far, a crucial question remains unanswered: Does a slight but sustained increased absorption of lead cause permanent or significant impairment in children who appear "asymptomatic?" (30)

In deciding whether or not children are symptomatic, one must, of course, bear in mind that lead poisoning presents no pathognomonic signs or symptoms in children. Early manifestations of central nervous system involvement, such as behavior changes, are often overlooked or misinterpreted. They may be recognized only in retrospect after frank encephalopathy and irreversible brain damage have occurred. Some parents who thought their children had no symptoms reported improved behavior and language ability following treatment. (30)

There is little doubt that acute, high-level lead exposure in young children can produce overt manifestations of neurotoxicity. Mental retardation is an established sequela of lead-induced encephalopathy in children. The extent and nature of lead-induced neurotoxicity following long-term low-level lead exposure during the developmental years is also of continuing interest. (29)

Some neuropathic effects have been observed for several cases at blood levels of 60 to 80 micrograms/dl, and in other cases peripheralneuropathy (disease around the periphery of the kidney) was associated with blood lead values of 30 micrograms/dl; however, in the latter cases, lead lines in long bones suggest probable past exposures leading to prior blood lead levels at least as high as 40 to 60 micrograms/dl (based on the data of Betts).[2] In each of the present case studies, there was reported some, if not complete, recovery of affected motor functions after treatment for lead poisoning. Further, it should be noted that a tentative association has been hypothesized between the existence of sickle cell disease and increased risk of peripheral neuropathy as a consequance of childhood lead exposure. Most of the cases reported involved inner-city black children, several with sickle cell trait.[2] In summary, it appears that (1) evidence for frank peripheral neuropathy in children certainly exists; (2) such neuropathy can be associated rather well with blood lead levels at least as low as 60 micrograms/dl; and (3) evidence suggests that inner-city children with sickle cell disease may be at special risk (29).

In a series of 102 cases of lead poisoning studied by Lilis, 18 cases of clinically verified chronic nephropathy were found (31). For the whole series, the mean blood lead level was approximately 80 micrograms/dl with a range of 42 to 141 micrograms/dl. Nephropathy was more common among patients who had been exposed to lead for more than 10 years. In both studies reduced urea clearance preceded reduced creatinine (a component of urine) clearance(31).

Data suggesting that lead exerts adverse effects on human reproductive functions have existed in the literature since before the turn of the century.[13] For example, Legge, in summarizing the reports of 11 English factory inspectors in 1897, found that of 212 pregnancies in 77 female lead workers only 61 living children were produced. Fifteen workers had never become pregnant. There were 21 stillborns, miscarriages occurred 90 times, and, of 101 children born, 40 died in the first year. Legge also noted that when pregnant animals were fed lead they always aborted. He concluded that maternal exposure to lead resulted in a direct action of the element on the fetus. (32)

In 1911, Oliver published statistics in Britain on the effect of lead on pregnancy which showed that the miscarriage rate was elevated among women employed in industries in which they were exposed to lead. (33)

In a more recent study of the pregnancies of 104 Japanese women married to lead workers before and after their husbands began lead work, miscarriages increased to 84.2/1000 pregancies from a prelead rate of 45.6/1000. The miscarriage rate for 75 women not exposed to lead was 59.1/1000. (34)

Another recent study by Fahim et al. in humans suggests that subtoxic lead absorption during pregnancy may be associated with an increased incidence of preterm delivery and early membrane rupture: 253 women deliviered, Missouri (Region I), which is 60 to 80 miles from lead smelters, and 249 women delivered in Columbia, Missouri (Region II), where there is no lead industry. The incidence of term pregnancies with early membrane rupture was 17 percent in Region I and 0.41 percent in Region II. The incidence of premature deliveries was 13.04 and 3 percent, respectively. A high correlation was found between lead concentrations in maternal and fetal blood: both were significantly higher in the cases of preterm pregnancies and early membrane ruptures than in term pregnancies. (35)

The above studies clearly demonstrate an adverse effect of lead on human reproductive functions, ranging from reduced pregnancy rates to increased incidence of miscarriages, premature deliveries, and stillbirths.(29)

Consistent relationships between blood lead levels and exposure to lead-containing soil have been shown. Also, children exposed to higher concentrations of soil and house dust lead have been shown to have elevated concentrations of lead on their hands. The intermediate link, from elevated hand levels of lead to elevated blood levels, has not yet been established.

Quantitatively, blood lead levels have been shown to increase 3 to 6 percent given a doubling of the soil or dust lead content. (29)

Significant water lead exposures in this country have only occurred in places having a soft water supply and using leaded pipes. Such exposures have been shown to be associated with significant elevations of blood lead. They have also been linked to cases of mental retardation. (29, 36)

Exposure to leaded paint still constitutes a very serious problem for American children in urban settings. Although new regulations of the lead content of paint should alleviate the problem in new housing, the poorly enforced regulation and lack of regulation of the past have left a heavy burden of lead exposures. Most of the studies on lead poisoning in children have assumed an association with leaded paint. It is very difficult in these studies to measure the actual amount of exposure. There is nevertheless incontrovertible evidence that the contribution from this source is very significant for certain segments of the population. (29)

Food lead exposures are thought to be a source of a significant portion of blood lead. Precise quantitative estimates of the relationship between food and blood lead are not available, however. Similarly, precise quantitative estimates are not available for the relative contributions of different sources to the total amount of lead in the diet. It is clear, however, that probably the largest proportion of dietary lead is derived from food processing (e.g., from solder in the seams of cans), and some is also derived from lead in the air and the soil. (29)

RECENT FINDINGS

Surveys conducted in the early 1970's indicated that the problem is not confined to large urban slums and areas east of the Mississippi River, as previously thought. One study of 14 cities with populations of 10,000 to 150,000 indicated that the magnitude of the problem there was comparable to that found in large cities. A DHEW survey of 52 communities throughout the nation revealed that undue lead absorption among children was geographically widespread. Screening data collected in the last five years confirmed this. Rural areas are not spared but the prevalence is lower there.

A few years ago the National Institute of Health estimated 400,000 children in the United States are affected by lead poisoning each year; of those 200 die and 800 have enough lead in their blood to produce permanent brain damage. (37)

In a report by the Center for Disease Control, the number of children identified with lead toxicity increased 41.1% over the number screened in the equivalent time period in 1978. The magnitude of the problem is demonstrated in the third quarter of the 1979 fiscal year whereas 7400 of the 126,300 children screened had lead toxicity. If this represents our national average, then approximately 6% of our nations' children may be suffering from lead poisoning. (38)

Children are more prone to have lead poisoning for the following reasons:

1. Their greater metabolic needs per kilogram body weight results in higher lead intake from food, liquid and inspired air.

2. Children are exposed to higher air lead levels. For example, the air lead concentration on Main Street in Brattleboro, Vermont was 3.96 micrograms/m^3 above 4 feet and 8.01 micrograms/m^3 below 4 feet.

3. "Pica" is indiscriminate eating of inappropriate or odd substances and is derived from the latin word, "magpie". The average house dust in Boston contains 1000 to 2000 ppm lead, and some areas reached concentrations of 20,000 ppm in 1973. In South Carolina the soil levels ranged from 1 to 12,000 ppm.

4. The blood brain barrier is not completely developed in children and probably explains why encephalopathy is rare in adults but is common in children with lead poisoning.

A survey to ascertain the prevalence of increased Pb absorption among children living near a primary Pb smelter in northern Idaho was taken among 1149 children 1 to 9 years old during August, 1974. Blood Pb samples were obtained in conjunction with a series of environmental samples, social and medical histories, and general observations of the home environment. (39)

The observed annual average ambient air lead concentration ranged from approximately 0.5–24,600 ppm. Blood lead levels of children (ages 1–9 years) are related to a host of environmental variables via regression techniques. Blood lead levels were found to be most influenced by five variables. These variables are: ambient air lead, soil lead, age of the child, dustiness of the home, and occupational status of the parents. It is concluded, based upon the results of this study, that any environmental control strategy should address both the air and the soil. Soil levels in excess of 1000 ppm lead as well as air lead levels greater than 2 micrograms Pb/m^3, 30 day average, were found to be unacceptable. (39)

In the August 1974 survey,99% of the 1 to 9 yr old children living within 1 mi of the smelter were found to have levels in excess of 40 micrograms Pb/100 ml. The frequency of abnormal Pb absorption was found to decrease with increasing distance from the smelter. Following the announcement of these results, certain emergency measures were inititated to reduce the risk of Pb intoxication. These measures included: (1) chelation of children over 80 micrograms Pb/100 ml, (2) purchase and destruction of as many homes as possible within 1/2 mi of the smelter, (3) distribution of "clean" soil and gravel to cover highly contaminated areas, (4) initiation of hygiene program in the schools, and (5) reduction of ambient air levels through reduction of smelter emissions. (39)

A follow-up study of 781 children was conducted in August 1975 to ascertain the change in blood Pb levels that may have resulted from altered environmental and social conditions. This follow-up survey revealed that the blood Pb levels had decreased. The mean Pb levels found in the ambient air and soil decreased with increasing distance from the smelter, following much the same pattern as that of blood levels. (39)

The nonspecificity of the symptoms of lead poisoning makes it difficult to diagnose. The diagnoses of the disease is particularly difficult in children when complaints of stomach ache, fatigue, weakness and irritability, vomiting, cramps, loss of appetite, pallor, convulsions or stupor. Finally, if enough lead accumulates in the body, the cerebral tissues soften and massive fluid accumultaion in the brain can cause death from acute encephalopathy.

As late as the summer of 1977, the symptoms of lead poisoning in a 2-year old boy living in New Jersey were missed during two visits (four days apart) to the emergency room of a hospital. Six weeks later the child was admitted to another hospital in a comatose state, following convulsive seizures at home. Although the child was immediately treated for lead encephalopathy, he remained in a coma for a month and was left with hemiparesis and questionable vision.

This child had a definite history of pica, his home was covered with lead-based paint and he had symptoms highly suggestive of lead poisoning - but no one considered the disease until it was too late, even though he lived in a city which had exerted considerable effort in public education and mass screening. His case emphasized the need for continuous viligance over a disease that will not easily disappear and the price we pay for not being watchful.

Subclinical neurophysiological effects may be observed among young children with moderately elevated blood levels, between 40 to 80 micrograms/100 dl. David observed higher lead levels and higher post penicillamine urine lead levels among the children with hyperactivity than in the control children. However, from this study, it could not be concluded whether lead exposure was caused by Hyperactivity or whether hyperactivity was caused by lead exposure.

It is not certain whether a Pb-B level in children below 100 micrograms/100 ml can be associated with an increase in prevalence of mental retardation or hyperactivity. Recently, Zielhuis concluded that there is some evidence that no-effect Pb-B levels for chronic encephalopathy are somewhat lower in children (probably at 50-60 micrograms/100 ml) than in adults (about 80 micrograms/100 ml). Since chronic effects on the central nervous system may be a consequence of repeated high exposures, it is difficult to evaluate dose-effect and dose-response relationships unless careful follow-up studies, including exact information on previous lead exposures, are performed.

Herbert L. Needleman has recently demonstrated that children with high lead levels scored significantly less well on the Wechsler Intelligence Scale for children (Revised) than those with low lead levels. This difference was also apparent on verbal subtests, on three other measures of auditory or speech processing and on a measure of attention. Also, lead exposure at doses below those producing symptoms severe enough to be diagnosed clinically, appear to be associated with neuropsychologic deficits that may interfere with classroom performance. (40)

Studies have shown that excess ambient air Pb levels can result in abnormal absorption in both children and adults. It seems that the mechanisms of absorption differ greatly between the two groups. Children represent the highest risk group and standard setting should be addressed to them. The total impact of ambient air contamination on children is due to both their physiological characteristics and habits. Normal childhood habits include the active ingestion of small amounts of soils and dust that contain particulate deposited by polluted air.

Any environmental control strategy devised for Pb should take into account all routes of exposure that significantly contribute to excess Pb absorption. The stratgey must not only address the lungs of the child, but the environment in which he lives. Five factors (air, soil, age of child, house dustiness, and parental occupation) have been identified as influencing blood Pb absorption. Two of those factors, air and soil contamination, can be regulated by environmental control. The other factors cannot be controlled by regulations. When setting a standard, a subjective decision must be made as to what percentage of the population should be allowed to exceed the 40 micrograms Pb/100 ml level. A second decision must be made as to what combination of air and soil limitations shall be applied to achieve that end. Based on the results of this study, it is apparent that soil levels in excess of 1000 ppm Pb of air levels in excess of 2 micrograms Pb/m^3, 30 days average, will result in excessive percentages of children exceeding the 40 micrograms Pb/100 ml blood Pb standard. (39)

Population at Risk

The concept of "special risk" is defined as a population segment exhibiting characteristics associated significantly higher probability of developing a condition , illness, or other abnormal status as a result of exposure to a given toxic agent. With respect to lead, two segments of human populations are currently definable: preschool children and unborn fetuses, with pregnant women as the recipient population of concern. These groups are of concern because of their high susceptibility (29).

Preschool Children

There is an impressive body of data that indicates that children are inherently more susceptible to lead by virtue of physiology and that they have a different relationship to exposure sources. These physiological factors include: (1) greater lead intake on a per-unit-body-weight basis; (2) greater net respiratory intake as well as greater net absorption and retention of lead entering the gastronintestinal tract; (3) rapid growth, which reduces the margin of safety against a variety of stresses, including nutritional deficiency; (4) certain incompletely developed defense mechanisms in very young children, such as blood-brain barrier in newborns; and (5) different partitioning of lead in the bones of children compared to that of adults, with only 60 to 65 percent of the lead body burden occurring in bone, and that fraction probably being more labile than in adults. (29)

Pregnant Women

Considerable evidence indicates that pregnant women are a population segment at risk with respect to lead mainly because of increased risk to the fetus and maternal complications. Lead crosses the placental barrier and does so at an early age in embryonic development. Although quantitative expressions of this risk connot be stated, certainly the potential for damage exists. (29)

Pregnancy places a physiolgical stress on a woman in terms of nutritional states that lead to iron and calcium deficiency, in consequence of which bone lead may be mobilized and the hematopoietic system may be placed at higher risk to lead exposure. In addition, data suggest that elevated blood lead levels in pregnant women may result in increased incidence of premature rupture, placing the mother as well as her newborn ina special risk category. (29)

United States Population in Relation to Probable Lead Exposures

With the exceception of those living in areas with primary lead smelters, most populations exposed to lead live in urban areas. Residents of the central city are at the highest risk in these urban areas. Therefore, for other than point stationary sources of lead, it is the urban population that is at risk, and in particular, central city residents. Blacks and Hispanics are probably subject to greater exposure to airborne lead because higher proportions of these segments of the population live in urban areas. In the United States in 1970, 149 million persons were reported to live in urban areas, and 64 million in the central cities. These figures include about 12 million children under 5 years of age, with approximately 5 million of these children living in central-city areas. There are an estimated 600,000 children in the United States with blood lead values greater than 40 micrograms/dl. These values do not result from exposure to airborne lead alone; they also include exposure to leaded paint and lead in food. In view of the narrow margin of safety from currently observed urban increment of lead from the air would be a great risk for adverse health effects. (29)

Of the roughly 3 million U.S. pregnancies per year, inner-city women are estimated to account for 500,000. In view of available data, these women and their unborn children are also at high risk. (29)

ECOLOGICAL EFFECTS (29)

As a natural constituent, lead does not usually pose a threat to ecosystems. The redistribution of naturally occurring lead in the environment, however, has now caused some concern that lead may represent a potential threat to the ecosystem. For example, studies have shown a fivefold increase in lead in tree rings during the last 50 years; this accumulation may serve as a useful index of patterns of environmental lead accumulation.

There are also documented effects of lead on domestic animals, wildlife, aquatic life. Lead poisoning in domestic animals produces varying degrees of derangement of the central nervous system, gastrointestinal tract, muscular system, and hematopoietic system. As is true in man, younger animals appear to be more sensitive than older ones.

Lead toxicity in aquatic organisms has been observed and studied experimentally. Symptoms of chronic lead poisoning in fish include anemia, possible damage to the respiratory system, growth inhibition, and retardation of sexual maturity.

There is evidence that lead has both harmful and beneficial effects on plants. Plants are exposed to lead through the leaves, stems, bark, or roots.

Effects on Invertebrates (42)

The effects of lead on invertebrates are significant because invertebrates are components of food chains and serve as an important food source for a large number of organisms which occupy higher trophic levels. The effects of lead vary from growth inhibition to death. Toxicity bioassays utilizing inorganic lead concentrations above the maximum allowable limit (0.05ppm) reveal a wide range of LC_{50} values which in many cases, depend on factors such as pH and hardness of water.

Lead nitrate (1 to 10 ppm) in natural seawater induced morphological changes and tissue reorganization of hydranths of the colonial coelenterate *Eirene vividuala*.

Lead chloride impairs reproduction and is lethal to the microcrustecean *Daphnia magna* at levels ranging 0.1 to 10 ppm.

Environmental lead levels of 0.1 and 0.2 ppm produced atrophy of gonadal tissue, and edema. The same lead levels were also very toxic to the oysters. For hard-shell clam embroyos, the LC_{50} for lead nitrate was 0.78 ppm as compared to 2.45 ppm for oyster embryos. The significance of any level of toxicity among those economically important invertebrates is that the success of their populations depends upon the success of the embryos in the environment.

Effects on Fishes (42)

Elevated lead concentrations are toxic or deleterious to fishes. Concentrations of lead and lead compounds that have been found to be lethal or toxic to fish under specific conditions, ranging from 0.1 mg/l to several thousand mg/l depending on species of fish, time of exposure, and type of water.

Embryo and fingerling stages of fish are most susceptible to lead toxicity. Highest mortality rates occurred among brook trout alevins upon exposures to lead nitrate. Embryos in the eggs of brook trout became severely deformed upon exposure of 500 ppb of lead. Similar deformations were noted to have occurred in fat head minnows at 240 ppb. The growth of fingerling brook trout was reduced at lead concentrations of 25 mg/liter per day administered as a slug dose.

Subtle effects result when fish are exposed to lead for entended periods of time. Catfish exposed to 5ppm lead acetate developed injured erythorocytes, progressive anemia, and a regenerative response in peripheral blood.

Rainbow trout, chronically exposed to lead nitrate concentrations up to 3.24 mg/l in hard water for 19 months, developed black tails, eroded caudal fins, and spiral curvatures.

The data presented show the lead upon acute or chronic exposures represent a hazard in terms of interfering with fish production. That lead interferes with embryonic development, the growth and development of young fish, and the behavior of fish is sufficient evidence to support the fact that lead in sufficient quantities (presumably levels greater than 0.05ppm or 50ppb) will cause a decreased population success for one of man's most important food sources.

Effects on Birds (42)

Lead poisoning among water fowl is characterized by typical signs and lesions as well as death.

Lead acetate was toxic to young Japanese quail at the level of 500 and 1000ppm. This toxicity was evidenced by an inhibition of normal growth and anemia. Additional data suggested that lead interfered with normal sexual development in male quail.

Mallard drakes exposed to particulate lead in four concentrations 178 grams per square meter in simulated marsh areas over a 14-week period developed atrophied livers, hydropericardia, and distended gallbladders.

Effects on Mammals (23)

Chronic lead poisoning in horses having fed on contaminated vegetation within the "fallout" area of a lead smelter was characterized by anonexia, loss of body weight, muscular weakness, anemia.

Chronic exposures to 6-7 mg of lead per kilogram of body weight (ppm) per day can result in toxicosis in cattle. Acute etyal, single exposures are 400 to 600 mg of lead per kilogram of body weight for adult cattle and 0.2 to 0.4 gram for lead (as lead acetate, carborate, or oxide) per kilogram

Incidents of lead poisoning in cattle and horses caused by emissions from stationary sources have been reported from Blackwell, Oklahoma; Benecia, California and several other areas.

The State of California Air Resources Board Coordinated a joint study, conducted by several state agencies between March 1970 and November 1971, to determine the cause of death of a number of horses in Benicis area from 1968 to 1970. The evidence strongly suggested that the horses died of lead poisoning that was caused by ingestion of lead deposited on pasture grass from a smelter plant at Selby, California. The ambient air concentrations were typical of those found in urban and surburban areas. It was concluded that horses in this area should not be allowed to subsist on pasture grass alone, but should receive supplemental feed. (29)

Hammond and Aronson have estimated that the minimal cumulative lethal dose of lead for a cow is 6 to 7 mg/kg of body weight per day. They state that this intake represents a concentration of about 300 ppm in the total diet. In cattle that consumed lead contaminated hay and corn silage grown in a field adjacent to a smelter, fatal lead poisoning occurred after approximately 2 months.

Horses are more susceptible than cattle to poisoning from the chronic intake of lead. In the Trail, B.C., study, horses grazing near a lead smelter developed overt symptoms of lead poisoning, but cows in the same pasture were not clinically affected. The minimal toxic dosage for a horse has been estimated to be between 1.7 and 2.4 mg/kg body weight daily, which is approximately 80 ppm dry weight in forage.

Horses sometimes, though infrequently, pull up plants and eat roots and soil along with leaves. This practice may be a factor in increasing their lead intake relative to that of cattle grazing the same pasture.

Lead poisoning in all domestic animals produces various degrees of derangement of the central nervous system, gastrointestinal tract, muscular system, and hematopoietic system. Younger animals appear to be more sensitive than older ones. Calves may suddenly begin to bellow and stagger about rolling their eyes, frothing at the mouth, and crashing blindly into objects. This phase may last up to 2 hours, after which a sudden collapse occurs. In less severe cases, depression, anorexia, and colic may be observed. The animals may become blind and may grind their teeth, move in a circle, push against objects, and lose their muscle coordination. Mature cattle display fewer overt symptoms, although the syndrome of maniacal excitement is not uncommon.

ENVIRONMENTAL CONTAMINATION

Soil

Several studies have identified Amarillo, Dallas, and El Paso, Texas and Bartlesville, Oklahoma as towns which have problems with lead contamination.

The facility in Amarillo which contributed to the lead problem has been recently closed but the soil contamination remains.

There are three lead smelters in Dallas. RSR is the largest and most modern of the Dallas smelters. Dixie and NL are the other two. Data from a soil survey conducted around the smelters in 1972 showed lead concentration in soil adjacent to the RSR smelter ranged from 3,940 to 5,520 ppm. Dixie soil levels ranged from 580 to 233,834 ppm and NL levels ranged from 1,620 to 464,000 ppm. (42)

In January 1972, a study was done to test the effects of prolonged subclinical elevations of lead in children in El Paso, Texas. Smeltertown, isolated between the Rio Grande River and a large smelter near El Paso had existed for some 80 years in a climate receiving approximately 6 inches of rain per year. There was no drainage system, no grass, dirt streets, dirt yards, and a few trees. Soil lead levels in the area averaged 3300 ppm. Presently, there is no direct threat to humans since Smeltertown was evacuated. Another study conducted in December, 1974 in the Juarez area showed soil levels ranging from 14 to 492 ppm. A definite correlation was established between the levels in blood and the concentration of lead in soil. (43, 45)

A recent (1979) follow-up epidemological study summarizes the present environmental lead situation in El Paso. (44)

The principal finding in this follow-up study was that blood lead levels in children living within 1.6 Km of the El Paso smeltery had decreased significantly from 1972 to 1977. The decline coincided with the installation of pollution controls at the smelter and paralled substantial declines in environmental lead levels. (44)

Data from this study indicates that mean blood lead levels were found to have decreased from 41.4 to 17.7/dl in children living within 0.8 Km of the plant and from 31.2 to 20.2 ug/dl in children living at 0.8 to 1.6 Km. (45)

There occurred a 15-fold reduction in the lead content of household dust within 1.6Km of the smeltery and a fourfold decrease in the mean lead concentration of topsoil. (44)

The lead problems in Bartlesville, Oklahoma has been controversial for several years. The National Zinc Company has a smelter there. The highest mean blood lead levels encountered in a nationwide survey of heavy metal absorption in children living near primary copper, lead and zinc smelters were in the children tested in Bartlesville. School grounds soil samples ranged from 26.3 to 1,705 ppm lead in 1979.

Water

The following table shows regional concentrations of lead in water.(46)

	1 No. of Stations	2 5% 50 ug/L	3 Max 150 ug/L
Regionwide	889	433 (49%)	156 (18%)
Arkansas	298	129 (43%)	66 (22%)
Louisiana	188	74 (39%)	25 (13%)
New Mexico	79	45 (57%)	20 (25%)
Oklahoma	244	133 (55%)	35 (14%)
Texas	80	52 (65%)	10 (13%)

Column 1 - Number of stations where samples have been analyzed for lead.

Column 2 - Number of stations in which 5% or more of the samples had concentrations greater than 50 ug/L.

Column 3 - Number of stations in which samples had concentrations greater than 150 ug/L.

Air

In the past eight years 1970 to 1978 the following cities in Region 6 have exceeded the lead air standard: Albuquerque, Baton Rouge, Dallas, El Paso, Houston, Little Rock, Oklahoma City, San Antonio and Shreveport. In more recent years 1975 - 1978, all of these same cities with the exception of Shreveport have still exceeded the air standards. The highest lead levels have been found in Oklahoma and Texas. (47)

Monitors located adjacent to the three smelters in Dallas recorded the following air levels from 1974 to 1978: (42)

PSR 5-100 ug/m^3, Dixie 14-220 ug/m^3, NL 40-400 ug/m^3

The neighborhood monitor located one mile from RSR recorded a range of 1.3 to 3.3 ug Pb/m^3 monthly (1.8 average). NL and Dixie are located in the same area approximately 1000' from another monitoring site. The lead concentrations ranged at this site from 3.5 - 13.0 ug Pb/m^3 monthly (8.6 average). Monitoring sites in neighborhoods without smelters averaged 0.32 - 2.66 ug Pb/m^3 (1.33 average). The monitoring site near the RSR smelter is not downwind of RSR, thus ambient concentration at RSR appears lower than at NL and Dixie.

TABLE I: Areas with Highest Air-Lead Values(1975)

URBAN (1975 yearly average $\mu g/m^3$)

City	$\mu g/m^3$ Lead
Burbank, California	2.97
Dallas, Texas	2.77
Chicago, Illinois	2.69
St. Paul, MN	2.15
Los Angelos, CA	2.12
Phoenix, AZ	2.08
Torrence, CA	2.05
Houston, Texas	1.91
Scranton, PN	1.89
Memphis, TN	1.82

Nonurban (very few sites selected, mainly one/state)

City	$\mu g/m^3$ Lead
Chicago area	2.19
Oklahoma City area (sites)	1.31
(sites)	0.98
King Co., Washington	0.45
Washington Co., Rhode Island	0.25

Source: NAMS Network Report

TABLE II LEAD-AIR DATA for REGION 6 - 1975

source: NAMS Annual Report

Location	Lead Concentration (ug/m³)				
	1st qtr.	2nd qtr.	3rd qtr.	4th qtr.	Average for year
ARKANSAS:					
El Dorado	*	*	*	*	-
Little Rock	0.78	0.86	0.80	0.91	0.84
Texarkana	0.60	0.34	*	0.38	0.44
W. Memphis 001	0.87	0.63	0.79	*	0.76
002	*	*	*	*	-
LOUISIANA:					
Baton Rouge	0.60	0.62	0.70	1.1	0.77
Iberville	0.19	*	0.22	*	-
New Orleans	1.0	0.75	0.75	0.90	0.86
Shreveport	0.42	0.37	0.81	0.83	0.61
NEW MEXICO:					
Albuquerque	*	0.28	*	*	-
OKLAHOMA:					
Cherokee	*	*	*	*	-
Tulsa	0.48	0.40	0.39	*	0.42
TEXAS:					
Amarillo	*	*	*	*	-
Austin	0.58	0.64	0.72	*	0.64
Beaumont	*	*	0.80	*	-
Corpus Christi	0.88	*	0.91	1.1	0.95
Dallas	2.9	2.3	3.0	2.9	2.8
El Paso	2.3	1.3	.69	2.0	1.6
Ft. Worth	0.60	0.43	0.95	.96	0.73
Houston	2.2	1.3	2.0	2.1	1.9
Lubbock	0.37	*	0.52	1.1	0.66
Pasadena	0.92	0.83	*	0.91	0.89
San Antonio	0.83	0.57	0.55	0.91	0.71
Wichita Falls	0.44	0.37	*	*	-

* Below detection limit

Principal IRLG Actions and Current Status

OSHA promulgated its occupational exposure standard on November 14, 1978 (43 FR 52952). Supplementary technical material supporting the standard appeared in the Federal Register on November 21, 1978 (43 FR 54353). On November 13, 1978, the Lead Industry Association and the Battery Council International petitioned for judicial review of the standard. The Court of Appeals for the District of Columbia issued a partial stay of the standard pending completion of its review.

EPA promulgated a national ambient air quality standard for lead on October 5, 1978 (43 FR 46246). The Lead Industry Association, in a separate suit, challenged EPA's standard. This case is not yet resolved.

FDA plans to set tolerances for all food; however, at present its priorities are on infant foods. A proposed tolerance level for evaporated milk should be finalized in Fall 1979. Currently, FDA is evaluating data from surveys of lead in infant food, and is continuing surveillance on and evaluation of the data for infant juices. FDA expects to publish the results of these surveys in 1979, after which an advance notice of proposed rulemaking will appear in the Federal Register. Regulation of other foods will begin after the infant food rules are promulgated.

On September 1, 1977, CPSC issued rules banning paint containing certain levels of lead and toys and furniture bearing such paint. In response to a petition, CPSC amended the rule on March 2, 1978 (43 FR 8514) to exempt metal furniture, except children's furniture bearing factory-applied coatings. Data presented to CPSC in the petition showed that factory-applied coatings from metal furniture do not chip or chalk and are therefore inaccessible for ingestion by children. The effective date of the amended rule was February 28, 1978 (the date the rule was signed).

EPA published summaries of both aquatic and health-based water quality criteria on March 15, 1979 (44 FR 15926). These criteria are not water quality standards and have no regulatory authority; however, they may form the basis for enforceable standards if adopted through rulemaking by a state or by EPA.

EPA may issue technical amendments to the existing drinking water regulations in March 1979. The amendments will not change the standards, but may change monitoring procedures. Eventually EPA expects to revise all of the existing drinking water regulations. The National Academy of Sciences has recommended that the standard, presently 50 ppb, be lowered. EPA proposes a study be done on corrosion of lead pipe distribution systems before revising its standard; however, no study has been initiated yet.

EPA is administering a phase-down program for lead in gasoline. The program stipulates a reduction to 0.3 grams/gallon by October.

By 1981 EPA expects to establish effluent guidelines. EPA is currently screening 36 categories (several categories were divided) for the presence of significant amounts of lead in the effluents. The agency plans to begin proposing guidelines in mid-1979.

On March 15, 1979, EPA published a notice announcing the availability for public comment of water quality criteria for lead. Comments are due by May 14, 1979. Final publication is planned for late in 1979.

In late 1979 EPA expects to propose a revised drinking water standard for lead.

On December 18, 1978, EPA proposed hazardous waste rules. Lead is listed as one of the hazardous contaminants in these rules. (See the Waste Disposal work plan for more details).

In October 1978, the task force on decorated glassware (FDA, EPA, and CPSC) gave its report to the agency principals. The report recommended that the agencies support a voluntary standard limiting leachable cadmium to 3.5 ppm and lead to 50 ppm on the lip and rim of the glassware, using the modified testing methods agreed upon. The task force also recommended that industry establish a monitoring program for cadmium and lead. The agency principals published the recommendations in the Federal Register on December 15, 1978 (43 FR 58633).

CURRENT CONTROLS - EPA

Lead emissions can be controlled through limitations in both air and water (National Pollution Discharge Elimination System [NPDES]) permits. Currently only fourteen facilities which we have identified as lead handling or emitting facilities, have an NPDES permit which contains a lead discharge limit (Table I). The other facilities listed in Appendix A and Tables I - IV do not have any water discharge limitations for lead. Air emissions are controlled by the States in Region 6 which have all been delegated authority under the Clean Air Act.

Under the Clean Water Act, the following industries have guidelines that include lead limits:

Electroplating
Chlorine, Sodium, or Potassium Hydroxide Products (see Table II)
Vacuum Degreasing
Hot Coating - Terne
Primary Copper Smelting
Primary Lead
Television Picture Tube
Hand Pressed and Blown Glass Manuf. (see Table III)
Latex Rubber (lead sheated hose products)
Medium-Sized General Mold Extruded (lead sheath)
Large-Sized General Mold Extruded (lead sheath)
Base and Precious Metals
Ferroalloy Ores

In addition, Storage Battery Manufacturing Facilities (see Table IV) will have guidelines for lead limits, but regulations covering this industry have not been promulgated.

Many of the above industries do not have lead limitations (Tables I-IV) in their NPDES permits. These industries should be carefully reviewed and if appropriate a lead limit should be included in their NPDES permits.

The National Pollutant Discharge Elimination System (NPDES) began in 1973. Most permits issued since that time have been issued for a maximum of five years. The second round of permits issued has therefore begun and has a new and different emphasis. Now, in addition to the conventional pollutants, the Clean Water Act now requires the control of toxic pollutants. EPA is currently developing the effluent standards for these toxic pollutants. Sources are classified as primary industries that include manufacturing, and chemical industries that have toxic discharges. Twenty-one such categories have been identified. The secondary industries are generally free of toxic discharges In the interim, eighteen month permits are generally being issued to primary industries. Also, as a permit is issued or re-issued, it contains a clause that allows it to be modified to include an effluent standard as they are promulgated.

The effluent standards are being developed on an industry-by-industry basis. In other words, the effluent standards for the iron and steel manufacturing category are being developed as a group for that industry rather than for example, a lead standard being developed to be used in all industry categories.

Many of the deadlines for effluent standard development have been slipped. It is therefore very difficult to determine when the lead standards will be promulgated and for which industries to insure the control described by this plan.

Air emissions are controlled by the States through the development of State Implementation Plans (SIPs) for lead. A summary of each State's SIP is provided below with the exception of New Mexico which has not filed its plan.

ARKANSAS

Arkansas does not have any facilities which meet EPA's guidelines for stationary sources. These guidelines require the State to control lead emissions of 5 tons or greater per year for the following facilities:

Primary Lead Smelters
Secondary Lead Smelters
Primary Copper Smelters
Lead Gasoline Additive Plants
Lead-Acid Storage Battery Manufacturing Plant (2000 or more batteries/day)

Emissions from other plants must be controlled if they emit greater than 25 tons per year of lead.

LOUISIANA

Louisiana contains two facilities which meet EPA guidelines, Ethyl Corp. and Schuylkill in Baton Rouge. Ten other facilities are listed by Louisiana in their SIP but these facilities do not meet EPA guidelines (Table V) for lead sources.

OKLAHOMA

Oklahoma has only one facility which meets EPA guidelines for lead sources. This facility, Prestolite Battery, is located in Oklahoma City. Prestolite emits 17.9 lbs. per hour of lead from four different emission points with only 6.05 lbs. per hour of lead being emited at any one time. This facility may cause the lead air standard to be exceeded.

TEXAS

Texas has eleven facilities which meet EPA guidelines for lead sources. These facilities are listed in Table VI. Six of these facilities may cause the lead air standard to be exceeded. These facilities are ESB, Inc., Dixie Metal Corp. and RSR Corp. in Dallas; ASARCO, Inc. in El Paso; Gould, Inc. in Frisco, and Houston Lead in Houston. A diagram showing the emission locations of these six facilities is provided as Appendix B. In all, the Texas Air Control Board (TACB) states there are forty-one (41) sources which may emit significant amounts of lead into the air. The additional sources are not listed by the TACB.

Source Contributions to Daily Lead
Uptake in Humans (23)

The following tables were obtained from a recent EPA contract study (to estimate the degree to which each major environmental source of lead exposure contributes to an individual's total daily lead uptake.

Certain assumptions were used to define the amount of each source material consumed per day. Where appropriate, reference man values are utilized for daily air and food consumption rates.

Table 1 - presents pulmonary and gastrointestinal absorption rates utilized in subsequent calculations - most of these figures represent average absorption values for inhaled or ingested lead, as reported in the literature.

Table 2 - presents the relative contribution from each route of exposure (i.e., air, food, drinking water, soil/dust, paint) to an individual's total daily uptake.

Table 3 - provides an example of the actual calculation sequence employed. Table 4 - presents the source contribution factors calculated for air, food and drinking water for the average male, based on the assumed sets of exposure conditions.

Table 5 - presents the factors for a pregnant female

Table 6 and 7 - presents the source contribution factors for the average three year-old child without pica and for the child with pica.

*It should be noted that these tables represent presumed average values, and do not account for instances in which as particular environmental source is high.

Table -1
Basic Assumptions Employed in the Calculation of Individual Source Contribution Factors

Basic Assumptions	Level	Remarks
• Reference Man:		
Adult consumes:	2.0 liters H_2O/day	- Daily intake as suggested by NAS (1977); conservative estimate, since all beverages assumed to be water, which has higher [Pb] than generic beverages (See Table 2-3)
	≈2200g food/day	- Approximate daily intake for 18-yr old in FDA total diet studies; comparable to Reference Man (ICRP, 1975)
	22.8 m³ air/day	- Assumes 8 hrs light work, 8 hrs nonoccupational, and 8 hrs resting (ICRP, 1975)
• Pregnant female:	2.0 liters H_2O/day 2200g food/day 21.1 m³ air/day	
Child consumes:	1.4 liters H_2O/day	- Conservative estimate for 2-yr old child, <2-yr old assumed to obtain liquid from foods alone
	≈1000g food/day	- Approximate daily intake for child from FDA total diet studies
	4.7 m³ air/day	- For 2-yr old; based on ICRP (1975) values for newborn, one-, 10-, and 18-yr olds
	100 mg soil and dust/day	- Conservative estimate for normal hand-to-mouth activity in children (Lepow et al., 1974)
	142 mg paint/day	- Conservative estimate for children with pica (NAS, 1976)
• Absorption Characteristics:		
Gastrointestinal		
Adult	10%	- See Table 3-2, generally recognized approximation
Child	50% for food and water	- See Table 3-2; (NAS, 1976; EPA, 1972a)
	30% for soil/dust	- Estimate based on compromise between food and paint absorption rates
	17%	- NAS, 1976
Pulmonary		
Adult/child	40%	- See Table 3-1, reasonable approximation for inhalation of ambient air; no attempt has been made to allocate inhaled particles, based on size to pulmonary (<=3μm) or gastrointestinal (>=3μm) absorption routes
Dermal	Insignificant	- Relatively unimportant, except in rare circumstances

Table -2

Representative Environmental Lead Exposure Levels

Exposure Routes		Remarks
Diet		
Adult (male)	208 µg/day	See Tables 2-4, 2-5, estimates based on reasonable range between diet preferences, excludes that contribution from beverages (including beverage contribution, 254 µg/day for adult, 115 for 3 yr old child; adult female diet approximately 80% that of males)
(female)	166 µg/day	
Child (3 yr old)	93 µg/day	
Ambient Air		
Urban average	0.69 µg/m^3	-See Table 2-1 for details; annual quarterly composite average NASN 1974 data
Non-Urban average	0.11 µg/m^3	
Ambient Air Standard	1.5 µg/m^3	-National ambient air quality standard (based on 90-day averaging)
Drinking Water	10 µg/l	
	25 µg/l	
	50 µg/l	-National interim primary drinking water standard
	100 µg/l	
Additional Exposure Routes for Children with Pica		
Paint	500 µg/g	-Current proposed standard
	8,000 µg/g	-Assumed mean, determined from existing house and market surveys (NAS, 1976; Bird, 1971)
Soil/dust (rural)	60 µg/g	Representative rural and urban values (Bethea and Bethea, 1975; Lepow et al., 1974; Rolfe and Haney, 1975)
(urban)	11,000 µg/g	

Table -3

Calculation Sequence in Determining Source Contribution Factors: Childhood Case

Source	Concentration	x	Consumption Rate	x	Absorption Rate	=	Daily Uptake in Children	Percent of Total Daily Uptake
Drinking Water	50 µg/l		1.4 liters/day		0.50		35.0 µg/day	5.8
Air	1.5 µg/m^3		4.7 m^3/day		0.40		2.8 µg/day	<1
Food	93 µg/day		—		0.50		46.5 µg/day	7.7
Soil/dust	11,000 µg/g		100 mg/day		0.30		330.0 µg/day	54.3
Paint	8,000 µg/g		142 µg/day		0.17		193 µg/day	31.8
					Total		607.3 µg/day	

Table 4
Estimated Daily Lead Uptake in Adult Males (µg/day)

		Rural			Urban			Ambient Air Standard		
	Source	Level	Uptake	Percent Contribution	Level	Uptake	Percent Contribution	Level	Uptake	Percent Contribution
Water @ 10 µg/l	Air	0.11 µg/m^3	1.0	4.2	0.89 µg/m^3	8.1	26.2	1.5 µg/m^3	13.7	37.5
	Food	208 µg/day	20.8	87.4	208 µg/day	20.8	67.3	208 µg/day	20.8	57.0
	Drinking Water	10 µg/l	2	8.4	10 µg/l	2	6.5	10 µg/l	2.0	5.5
	Total		23.8			30.9			36.5	
Water @ 25 µg/l	Air	0.11 µg/m^3	1.0	3.7	0.89 µg/m^3	8.1	23.8	1.5 µg/m^3	13.7	34.7
	Food	208 µg/day	20.8	77.6	208 µg/day	20.8	61.4	208 µg/day	20.8	52.6
	Drinking Water	25 µg/l	5.0	18.7	25 µg/l	5.0	14.7	25 µg/l	5.0	12.7
	Total		26.8			33.9			39.5	
Water @ 50 µg/l	Air	0.11 µg/m^3	1.0	3.2	0.89 µg/m^3	8.1	20.8	1.5 µg/m^3	13.7	30.8
	Food	208 µg/day	20.8	65.4	208 µg/day	20.8	53.5	208 µg/day	20.8	46.7
	Drinking Water	50 µg/l	10.0	31.4	50 µg/l	10.0	25.7	50 µg/l	10.0	22.5
	Total		31.8			38.9			44.5	
Water @ 100 µg/l	Air	0.11 µg/m^3	1.0	2.4	0.89 µg/m^3	8.1	16.6	1.5 µg/m^3	13.7	25.1
	Food	208 µg/day	20.8	49.8	208 µg/day	20.8	42.5	208 µg/day	20.8	38.2
	Drinking Water	100 µg/l	20.0	47.8	100 µg/l	20.0	40.9	100 µg/l	20.0	36.7
	Total		41.8			48.9			54.5	
Water @ 200 µg/l	Air	0.11 µg/m^3	1.0	1.6	0.89 µg/m^3	8.1	11.8	1.5 µg/m^3	13.7	18.4
	Food	208 µg/day	20.8	33.7	208 µg/day	20.8	30.2	208 µg/day	20.8	27.9
	Drinking Water	200 µg/l	40.0	64.7	200 µg/l	40.0	58.0	200 µg/l	40.0	53.7
	Total		61.8			68.9			74.5	

Table -5
Estimated Daily Lead Uptake in Pregnant Females (µg/day)

		Rural			Urban			Ambient Air Standard		
	Source	Level	Uptake	Percent Contribution	Level	Uptake	Percent Contribution	Level	Uptake	Percent Contribution
Water @ 10 µg/l	Air	0.11 µg/m³	0.9	4.6	0.89 µg/m³	7.5	28.7	1.5 µg/m³	12.7	40.5
	Food	166 µg/day	16.6	85.1	166 µg/day	16.6	63.6	166 µg/day	16.6	53.1
	Drinking Water	10 µg/l	2.0	10.2	10 µg/l	2.0	7.7	10 µg/l	2.0	6.4
	Total		19.5			26.1			31.3	
Water @ 25 µg/l	Air	0.11 µg/m³	0.9	4.0	0.89 µg/m³	7.5	25.8	1.5 µg/m³	12.7	37.0
	Food	166 µg/day	16.6	73.8	166 µg/day	16.6	57.0	166 µg/day	16.6	48.4
	Drinking Water	25 µg/l	5.0	22.2	20 µg/l	5.0	17.2	25 µg/l	5.0	14.6
	Total		22.5			29.1			34.3	
Water @ 50 µg/l	Air	0.11 µg/m³	0.9	3.3	0.89 µg/m³	7.5	22.0	1.5 µg/m³	12.7	32.2
	Food	166 µg/day	16.6	60.4	166 µg/day	16.6	48.7	166 µg/day	16.6	42.3
	Drinking Water	50 µg/l	10.0	36.3	50 µg/l	10.0	29.3	50 µg/l	10.0	25.5
	Total		27.5			34.1			39.3	
Water @ 100 µg/l	Air	0.11 µg/m³	0.9	2.4	0.89 µg/m³	7.5	17.0	1.5 µg/m³	12.7	25.7
	Food	166 µg/day	16.6	44.3	166 µg/day	16.6	37.6	166 µg/day	16.6	33.7
	Drinking Water	100 µg/l	20.0	53.3	100 µg/l	20.0	45.4	100 µg/l	20.0	40.6
	Total		37.5			44.1			49.3	
Water @ 200 µg/l	Air	0.11 µg/m³	0.9	1.6	0.89 µg/m³	7.5	11.7	1.5 µg/m³	12.7	18.3
	Food	166 µg/day	16.6	28.9	166 µg/day	16.6	25.9	166 µg/day	16.6	24.0
	Drinking Water	200 µg/l	40.0	69.5	200 µg/l	40.0	62.4	200 µg/l	40.0	57.7
	Total		57.5			64.1			69.3	

Table -6
Estimated Daily Lead Uptake in Children Without Pica (µg/day)

		Rural			Urban			Ambient Air Standard		
	Source	Level	Uptake	Percent Contribution	Level	Uptake	Percent Contribution	Level	Uptake	Percent Contribution
Water @ 10 µg/l	Air	0.11 µg/m³	0.2	<1	0.89 µg/m³	1.7	<1	1.5 µg/m³	2.8	<1
	Food	93 µg/day	46.5	83.8	93 µg/day	46.5	12.1	93 µg/day	46.5	12.0
	Soil/Dust	60 µg/g	1.8	3.2	11000 µg/g	330	85.7	11000 µg/g	330	85.4
	Drinking Water	10 µg/l	7	12.6	10µg/l	7	1.8	10µg/l	7	1.8
	Total		55.5			385.2			386.3	
Water @ 25 µg/l	Air	0.11 µg/m³	0.2	<1	0.89 µg/m³	1.7	<1	1.5 µg/m³	2.8	<1
	Food	93 µg/day	46.5	70.5	93 µg/day	46.5	11.8	93 µg/day	46.5	11.7
	Soil/Dust	60 µg/g	1.8	2.7	11000 µg/g	330	83.4	11000 µg/g	330	83.2
	Drinking Water	25 µg/l	17.5	26.5	25 µg/l	17.5	4.4	25 µg/l	17.5	4.4
	Total		66.0			395.7			396.8	
Water @ 50 µg/l	Air	0.11 µg/m³	0.2	<1	0.89 µg/m³	1.7	<1	1.5 µg/m³	2.8	<1
	Food	93 µg/day	46.5	55.7	93 µg/day	46.5	11.3	93 µg/day	46.5	11.2
	Soil/Dust	60 µg/g	1.8	2.2	11000 µg/g	330	79.9	11000 µg/g	330	79.7
	Drinking Water	50 µg/l	35	41.9	50 µg/l	35	8.5	50 µg/l	35	8.4
	Total		83.5			413.2			414.3	
Water @ 100 µg/l	Air	0.11 µg/m³	0.2	<1	0.89 µg/m³	1.7	<1	1.5 µg/m³	2.8	<1
	Food	93 µg/day	46.5	39.2	93 µg/day	46.5	10.4	93 µg/day	46.5	10.3
	Soil/Dust	60 µg/g	1.8	1.5	11000 µg/g	330	73.6	11000 µg/g	330	73.4
	Drinking Water	100 µg/l	70	59	100 µg/l	70	15.6	100 µg/l	70	15.6
	Total		118.5			448.2			449.3	
Water @ 200 µg/l	Air	0.11 µg/m³	0.2	<1	0.79 µg/m³	1.7	<1	1.5 µg/m³	2.8	<1
	Food	93 µg/day	46.5	24.6	93 µg/day	46.5	9.0	93 µg/day	46.5	9.0
	Soil/Dust	60 µg/g	1.8	1.0	11000 µg/g	330	63.7	11000 µg/g	330	63.5
	Drinking Water	200 µg/l	140	74.2	200 µg/l	140	27.0	200 µg/l	140	27.0
	Total		188.5			518.2			519.3	

Table -7
Estimated Daily Lead Uptake in Children with Pica for Paint (µg/day)

		Rural					Urban					Ambient Air Standard				
			Uptake	Percent Contribution	Uptake	Percent Contribution		Uptake	Percent Contribution	Uptake	Percent Contribution		Uptake	Percent Contribution	Uptake	Percent Contribution
	Source	Level	Paint @ 800 µg/g		Paint @ 8000 µg/g		Level	Paint @ 800 µg/g		Paint @ 8000 µg/g		Level	Paint @ 800 µg/g		Paint @ 8000 µg/g	
Water @ 10 µg/l	Air	0.11 µg/m³	0.2	<1	0.2	<1	0.88 µg/m³	1.7	<1	1.7	<1	1.5 µg/m³	2.8	<1	2.8	<1
	Food	83 µg/day	46.5	66.3	46.5	18.6	83 µg/day	46.5	11.6	46.5	8.0	83 µg/day	46.5	11.6	46.5	8.0
	Soil/Dust	80 µg/g	1.8	2.6	1.8	<1	11000 µg/g	330	82.5	330	56.9	11000 µg/g	330	82.3	330	56.8
	Paint		14.6	20.8	194.3	77.8		14.6	3.7	194.3	33.5		14.6	3.6	194.3	33.5
	Drinking Water	10 µg/l	7	10.0	7	2.8	10 µg/l	7	1.8	7	1.2	10 µg/l	7.0	1.7	7.0	1.2
	Total		70.1		249.8			399.8		579.5			400.9		580.6	
Water @ 25 µg/l	Air	0.11 µg/m³	0.2	<1	0.2	<	0.88 µg/m³	1.7	<1	1.7	<1	1.5 µg/m³	2.8	<1	2.8	<1
	Food	83 µg/day	46.5	57.7	46.5	17.9	83 µg/day	46.5	11.3	46.5	7.9	83 µg/day	46.5	11.3	46.5	7.9
	Soil/Dust	80 µg/g	1.8	2.2	1.8	<1	11000 µg/g	330	80.4	330	55.9	11000 µg/g	330	80.2	330	55.8
	Paint		14.6	18.1	194.3	74.6		14.6	3.6	194.3	32.9		14.6	3.5	194.3	32.9
	Drinking Water	25 µg/l	17.5	21.7	17.5	6.7	25 µg/l	17.5	4.3	17.5	3.0	25 µg/l	17.5	4.3	17.5	3.0
	Total		80.6		260.3			410.3		590.0			411.4		591.1	
Water @ 50 µg/l	Air	0.11 µg/m³	0.2	<1	0.2	<1	0.88 µg/m³	1.7	<1	1.7	<1	1.5 µg/m³	2.8	<1	2.8	<1
	Food	83 µg/day	46.5	47.4	46.5	16.7	83 µg/day	46.5	10.9	46.5	7.7	83 µg/day	46.5	10.8	46.5	7.6
	Soil/Dust	80 µg/g	1.8	1.8	1.8	<1	11000 µg/g	330	77.1	330	54.3	11000 µg/g	330	76.9	330	54.2
	Paint		14.6	14.9	194.3	69.9		14.6	3.4	194.3	32.0		14.6	3.4	194.3	31.9
	Drinking Water	50 µg/l	35.0	35.6	35.0	12.6	50 µg/l	35.0	8.2	35.0	5.8	50 µg/l	35.0	8.2	35.0	5.8
	Total		98.1		277.8			427.8		607.5			428.9		608.6	
Water @ 100 µg/l	Air	0.11 µg/m³	0.2	<1	0.2	<1	0.88 µg/m³	1.7	<1	1.7	<1	1.5 µg/m³	2.8	<1	2.8	<1
	Food	83 µg/day	46.5	34.9	46.5	14.9	83 µg/day	46.5	10.0	46.5	7.2	83 µg/day	46.5	10.0	46.5	7.2
	Soil/Dust	80 µg/g	1.8	1.4	1.8	<1	11000 µg/g	330	71.3	330	51.4	11000 µg/g	330	71.1	330	51.3
	Paint		14.6	11.0	194.3	62.1		14.6	3.2	194.3	30.2		14.6	3.1	194.3	30.2
	Drinking Water	100 µg/l	70.0	52.6	70.0	22.4	100 µg/l	70.0	15.1	70.0	10.9	100 µg/l	70.0	15.1	70.0	10.9
	Total		133.1		312.8			462.8		642.5			463.9		643.6	
Water @ 200 µg/l	Air	0.11 µg/m³	0.2	<1	0.2	<1	0.88 µg/m³	1.7	<1	1.7	<1	1.5 µg/m³	2.8	<1	2.8	<1
	Food	83 µg/day	46.5	22.9	46.5	12.1	83 µg/day	46.5	8.7	46.5	6.5	83 µg/day	46.5	8.7	46.5	6.5
	Soil/Dust	80 µg/g	1.8	<1	1.8	<1	11000 µg/g	330	61.9	330	46.3	11000 µg/g	330	61.8	330	46.2
	Paint		14.6	7.2	194.3	50.8		14.6	2.7	194.3	27.3		14.6	2.7	194.3	27.2
	Drinking Water	200 µg/l	140	68.9	140	36.6	200 µg/l	140	26.3	140	19.6	200 µg/l	140	26.2	140	19.6
	Total		203.1		382.8			532.8		712.5			533.9		713.6	

Conclusions

The toxicological impact of lead is the cumulative result of exposure from many sources. Adverse health effects may result from continuous low-level exposure from several sources in ambient environment. Toxic effects are essentially due to the mobile fraction of absorbed lead within the body. This mobile fraction is composed of lead from any recent exposure, as well as the background level of easily mobilized lead previously deposited in the soft tissues and soft (trabecular) bone, and to a lesser extent, to that portion mobilized from dense bone.

Two subgroups within the general population have been identified as being more sensitive and at a greater risk to environmental lead exposure. Due to many factors, children under the age of four are especially susceptible to the toxic effects of lead. The fetus has also been identified as a lead sensitive individual, due to the immature state of development of certain organs. Because transplacental absorption is the major source of prenatal lead exposure, the pregnant female must be recognized as the exposure vehicle for the fetus. The blood-lead level in the fetus is approximately the same as that in the mother.

The Center for Disease Control (CDC) set the blood level of significant danger to children at 30 µg/dl. The level of 30 µg/dl set by the CDC is endorsed by the American Academy of Pediatrics and is now the target level defined by EPA as the level of undue lead exposure.

As the study by Drill (23) indicates there are appreciable differences in exposure conditions between specific subsets of the population (e.g., adults vs. children, children with pica vs. children without pica, rural vs. urban populations) and these can be characterized by average exposure assumptions for subpopulations in question.

This variability in exposure levels implies that there is considerable variability in the source contribution factors for all the environmental lead exposure sources.

If all sources of environmental lead are considered, the most effective means of reducing the overall blood-lead value of a given population can be determined. The model used in Drill's (23) study shows which environmental sources are most responsible for the given blood-lead level and thus identifies those areas in which regulatory action will have the greatest impact on the blood levels.

Therefore, it seems logical to approach the lead control from a sensitive populations perspective in localized situations.

Recent studies (EPA 600/2-79-039, 087, 093) on measurements of pollutants found in waste streams conducted for several industries: secondary lead and battery manufacturing, primary aluminum production and primary zinc production facilities, respectively, had removal efficiencies of 95% - 100% for lead upon treatment.

Deficiencies in Present Programs

1. No complete materials balance appears to have ever been made for lead.

   Estimated air emissions are probably the most completely identified and quantified. The EPA draft report covers most of the principal types of emission sources for which estimates are provided.

   There appears to be no comparable estimate of water emissions. Part of the problem here is that much, if not most, of the lead entering the hydrosphere, was originally an air emission, and double counting is a problem not amendable to an easy solution.

2. Only one state in Region 6 has developed a Water Quality criteria for lead.

   However, as compliance with effluent guidelines and other waste disposal regulations improves, lead concentrations in water should decrease.

3. The NASN(now referred to as Federal Reference) sampling protocol defines the total suspended particulate (TSP) material in ambient air when is comprised primarily of particles with diameters less than 1μ.

   A large portion of lead containing particles near smelters are in the 7.0μ size range (75%). These larger particles are more apt to settle out of the atmosphere quickly. Therefore, they contribute significantly to the lead levels in soil, dust, water and vegetation in the vicinity of the source and are not reflected by the particulate monitors because TSP sampling sites are usually located on rooftops for determining concentrations of total particulates and do not accurately depicit the situation at street level.

   Consequently, higher ambient lead levels are found with primary smelting operations located in the vicinity.

   The manufacture of lead containing antiknock compounds involves emissions to the atmosphere of an estimated 1,300 meteric tons contained lead.

   The losses of lead in wastewater from lead alkyl manufacturing plants was estimated at 60 metric tons.

   Losses also occur at the refinery, at the transfer terminals, at the bulk stations and at the service stations.

   Decline in use of leaded gas will cause a major decline in the loss of lead alkyls.

   Reinforcing this trend will be the EPA regulations on vapor recovery, designed to control hydrocarbon emissions.

4. It has been estimated that from 1 to 5 percent of lead deposited on roads is resuspended by passing mototist. 65 percent of lead in the air from 10-530 meters (30 to 1750 feet) from a well travelled highway consisted of particles under 2 µ and over 85 percent consisted of particles under 4 µ in diameter.

   Air-lead levels in houses adjacent to heavily traveled roads are almost identical with outside street air levels (particularly those houses without air conditioning).

   Urban atmospheres contain higher lead concentrations than nonurban atmospheres.

   The nature of automobile emissions are markedly influenced by the age of the vehicle, (i.e., both quantity and size increases with mileage).

5. Data from NASN represent a total of only 92 lead sampling sites, a small number on which to base either a regional or national average and the results should be used with caution.

6. Smelters still constitute local atmospheric lead problems, since most of the problem sites, being 75 or more years old, do not fall under New Source Performance Standards (NSPS) limitations and some other mechanism to control must be applied (   ).

7. Because of the diversity and lower grades of the feeds of secondary lead recovery operations, and a probably lower level of effective pollution control, the estimated emissions of lead to the atmosphere (755 metric tons) and the amount of lead wasted to land disposal (3,260 metric tons) exceed those of the primary lead smelting industry (   ).

8. It is estimated that the majority of scrap operations are sources of acid runoff and drainage which is saturated with lead. These operations constitute a diffuse and unassessed segment of industrial operations. They are estimated to account for a portion of the release of lead in the flows of sewage and drainage from municipal - industrial complexes.

9. Storage battery manufacturing operations result in annual losses of lead estimated as 22 metric tons to the air, 0.2 to 340 metric tons in wastewaters and 39 metric tons of lead contained in wastes destured for land disposal.

10. Emissions of lead to the atmosphere from Municipal Solid Waste incineration have been estimated to be 1,630 metric tons.

11. The contribution of lead pipes to the level of lead in drinking water can be significant.

    Bases on a 1974 EPA investigation of the lead levels in drinking H_2O, it was clean that with prolonged contact with the sections of lead pipe, lead content of the water reached undesirable levels, Almost 50% of the first drawn samples in the morning exceeded the 50 µg/l standard and the mean sample contained over 100 µg/l.

    Boston acidic water-steps were taken to adjust the pH of the water to reduce impact on lead pipes. (2,000 tons NaOH) This probably would not be a problem any where in Region 6 due to the age of our cities and the advent of cast iron pipe.

Rationale and Implementation Plan

In this plan we are concerned about protecting those children, pregnant mothers and fetuses who reside in the vicinity of industrial operations where the ambient levels may exceed the National Standard.

The National Ambient Air Quality Standard for lead makes the assumption based on Models that facilities with lead emissions of less than 5 tons per year will not cause the 1.5 ug/m^3 lead standard to be exceeded on a nationwide basis. However, in the Preamble to this Standard the Agency recognized the possibility of significant problems in the attainment of the Standard in the vicinity of non-ferrous smelters and other large industrial sources of lead emissions. It also recognized the potential need for establishing more than two (2) monitoring sites in an area. Therefore, the Standard includes the flexibility to allow Regional or State action to establish additional monitoring sites to adequately determine emission levels and to assess alternative engineering controls.

Accordingly, in this plan we will validate the National assumptions for localized situations.

In addition, none of the existing National Media Programs address soil as a major source of exposure for sensitive sub-groups of the population. Since a recent exposure model has identified soil as the major contributor to urban childhood we will focus out attention on this source.

Since the toxicological impact of lead is the cumulative result of exposure from all sources, i.e., air, water, food, soil/dust and etc., we are interested in instituting controls on those sources that will reduce the exposure potential for the most susceptible sub-groups in the populations.

Our primary focus will be on those sources which are not presently controlled, and identified as making the greatest contribution to the exposure of these sub-groups.

Secondary lead smelters located near residential areas contribute to air, soil/dust, water and vegetation exposure pathways and hence will constitute our priority source for action. The soil/dust lead contamination results in continuous exposures, even after the lead facility has ceased operation, through re-suspension and as such will also be included in our top priority efforts.

Federal regulations require the state to report to EPA all stationary sources that emit five tones of lead per year (t/yr) or more as a part of their SIP. These sources are referred to as "point sources". Submitting only the required information for point sources may be inadequate since some sources that emit less than 5 tons/yr have the potential to violate the National Ambient Air Quality Standard (NAAQS) for lead.

determined by the use of atmospheric dispersion modeling. The Supplementary Guidelines for Lead Implementation Plans (EPA-459/2-78-038) contains a detailed discussion of the procedures used to determine these values. Note from the Table that grey iron foundries and ferroalloy plants can exceed the NAAQS with only a 2 ton/year emission rate. If only the required point sources are reported, such sources as these will not be identified in the SIP's.

In view of the fact that urban residential areas located near smelters are also usually in old deteriorating sections of the community, it is highly probable that the houses could have lead pipes to carry water from the main to the house which could result in drinking water with lead levels above the 50 ug/l water quality standard. Therefore, the type of pipes in use in these areas and tap water lead levels will be investigated. Many families living in some of these areas will catch and eat fish from small creeks or ponds near by which collects the runoff from the contaminated soil. Such fish will probably exceed the FDA guideline.

Oklahoma is the only State in Region 6 with an established Water Quality Standard, therefore steps will be taken to encourage the establishment of such a standard in the other four states.

7. Implementation Plan

TASK A

A monitoring contract to identify level and extent of lead contamination in soil in residential and school playground areas.

a) Award contract by May 30, 1980

b) Review interim report by July 30, 1980

c) Final report due September 30, 1980

d) Evaluate final report by October 30, 1980

e) Provide local soil/dust data and evaluation to appropriate state and local agencies for follow-ups on November 15, 1980

f) Request state response of Proposed Action by January 30, 1981

g) Develop Federal Plan for addressing soil/dust problems in communities where state or local agencies fail to take appropriate action by February 30, 1981

TASK A - MANAGEMENT

Pesticides & Toxic Substances Branch will be responsible for the direction of this task with assistance from S & A and Enforcement Divisions.

LEVEL OF EFFORT

Fifty thousand dollars ($50,000) is available for negotiating this contract.

TASK 8

Establish additional air monitoring sites in neighborhoods located within two (2) miles of smelters.

a) Request additional existing data from local and state agencies by April 15, 1980

b) Receive and review replies from state by April 30, 1980

c) Evaluate data submitted by states and finalize workscope for Level of Effort (LOE) Air Monitoring Contract by May 15, 1980

d) Award LOE contract by May 30, 1980, (contingent on date HQTRS set-up LOE contract)

e) If LOE contract is not available, initiate 80A contract by July 30, 1980

f) Evaluate final report by November 30, 1980

g) Assess air emissions and control measures by December 30, 1980

h) Initiate action to require appropriate controls

TASK 8 - MANAGEMENT

Air Program Branch will provide funds for contract.

Toxic Substances staff will evaluate data, prepare workscope and administer contract with assistance from S & A Enforcement Divisions.

LEVEL OF EFFORT

About two (2) workyears and up to $100,000 in contract funds.

PERIOD OF PERFORMANCE

Data collection will begin immediately. The program will be completed when all the lead SIP's have been approved.

TASK C

Examine lead facilities in areas near residential communities and determine lead materials balance.

a) Request the names and locations of all the stationary sources of lead that emit 1-5 tons per year from the appropriate states to be submitted by June 15, 1980

b) Evaluate these sources and identify those with potential to exceed the NAAQS for lead by July 30, 1980

c) Air Programs Branch, will then require that the potential violations are included in the SIP Monitoring Programs by September 30, 1980

TASK C - MANAGEMENT

The Implementation Plan Section, Air Programs Branch

LEVEL OF EFFORT

A maximum of 160 hours will be expended.

PERIOD OF PERFORMANCE

Data collection will begin immediately. The program will be completed when all the lead SIP's have been approved.

TASK D

Encourage the establishment of Water Quality Standards in Arkansas, Louisiana, New Mexico and Texas.

a) Assess stream monitoring data for lead by May 30, 1980

b) Arrive at proposed instream standard(s) for lead by July 30, 1980

c) Send letters to states identifying extent of problem, proposing instream standard, and requesting state adoption, by September 30, 1980

d) Follow-up work by Water Program staff. Dates of state adoption and submittal will be largely out of our control. Estimate this will take 3-12 months, depending on state.

e) Incorporate identity of lead sources in 208 Water Quality Management Plans and evaluate controls by October 30, 1980

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*Louisiana has already submitted such information. New Mexico's SIP was not due until March 31, 1980; supplementary information can be requested.

TASK D - MANAGEMENT

Water Division, Water Quality staff will have the lead with assistance from S & A in the direction of this task.

TASK E

Conduct tap water sampling for lead levels in selected old neighborhood to determine if lead pipes are contributing to children's lead exposure.

a) This task will be included in workscope of contract in TASK A.

b) Results will be evaluated and provided to state and local agencies.

TASK E - MANAGEMENT

Toxics staff with the coordination of Water Supply staff in Water Division will be responsible for the Administration of the contract.

Water Supply Branch will be responsible for evaluation of data and initiation of appropriate action.

CHEMICAL WORKPLAN FOR LEAD

TASKS	MONTHS									
	April	May	June	July	Aug	Sept	Oct	Nov	Dec	Jan
A - Soil/Dust Monitoring										
-Award Contract		▲								
-Final Report				△		△	▲			
-Provide data to State/Local								△		
-Develop Federal Plan										△
B - Establish Air Monitoring Sites										
-Request State/Local data	△									
-Evaluate Data		△								
-Finalize Workscope (LOE)		△								
-Award LOE Contract?		△								
-Initiate BOA Contract				△						
-Evaluate Final Report								△		
-Assess Air Emissions									△	
C - Determine Lead Materials Balance										
-Request list of Sources			△							
-Evaluate Sources for compliance with NAAQS				△						
-Require SIP Revisions						△				
D - Establish WQSTD										
-Assess stream data		△								
-Propose standards				△						
-Letter to States requesting adoption						△				
E - Tap Water Sampling										
-Task included in TASK A Contract		△				▲				
-Provide data to State/Locals								△		

TABLE 1: STATIONARY SOURCE QUARTERLY MODELING RESULTS AND POINT SOURCE DEFINITION FOR NAAQS QUARTERLY AVERAGE OF 1.5 μg/m^3

Industry Type	Point Source* Definition (ton/yr)
Primary Lead	65
Primary Copper	54
Tetraethyl Lead Mfg.	29
Secondary Lead	6
Grey Iron	2
Ferroalloy	2
Battery Manufacture (500 BPD) w/o PbO production	4
Battery Manufacture (6500 BPD) w/PbO production	7

*Emission rates above which the NAAQS will be violated.

TABLE I

COMPANIES WITH NPDES PERMITS CONTAINING LEAD LIMITS

FACILITY	LEAD LIMIIT (lbs/day)		NORMAL DISCHARGE[1]	1979[2]
	Daily Avg.	Daily Max	lbs/day	Violations
Allied Chemical Baton Rouge, LA	5	10	4.18	2
ASARCO Corpus Christi, TX	1.61	3.22	2.9	4
BASF Wyandotte Geismar, LA	5.1	10.2	1.83	0
Ethyl Corp. Baton Rouge, LA	53.7	108	47.75	0
Ethyl Corp. Pasadena, TX	16	32	10.2	0
Dow Chemical Deer Park, TX	4.8	9.5	<2.42[3]	0
Gould, Inc. Shreveport, LA	0.15	0.35	0.14	3
Hooker Chem. & Plastics Hahnville, LA (Taft)	5	10	.3	0
Kaiser Aluminum Gramercy, LA	3.0	6.0	2.7	0
Nalco Chemical Freeport, TX	10	25	6.76	0
PPG Industries Corpus Christi, TX	5.1	10.1	3.7	4
Red Diamond Battery Hot Springs, AR	0.025	0.05	0 since7/78	0
Remington Arms Lonoke AR	0.74	1.5	0.13	0
Tyler Pipe Tyler, TX	1.9	3.4	0.8	0

1. Computed for six months period
2. Monthly violations during a six month period
3. Normal discharge based on (2) two months only. Dow did not discharge the other 4 months
4. Normal discharge based on single month. No other lead discharge in six month period

TABLE II

CHLORINE, SODIUM, OR POTASIUM HYDROXIDE PRODUCTS

COMPANY	LOCATION	NPDES PERMIT	LEAD LIMIT
Air Products	La Porte, TX	Yes	No
Allied Chemical	Baton Rouge, LA	Yes	Yes
Aluminum Co.of America	Point Comfort, TX	Yes	No
American Magnesium	Snyder, TX	Yes	No
BASF Wyandotte	Geismar, LA	Yes	Yes
Champion Intermed.	Pasadena, TX	No	-
Chromalloy	Houston, TX	No	-
Diamond Shamrock	Deer Park, TX	Yes	No
	La Porte, TX	No	-
Dow Chem. USA	Deer Park, TX	Yes	Yes
	Freeport, TX	Yes	No
	Plaquemine, LA	No	-
Ethyl Corp.*	Baton Rouge, LA	Yes	Yes
*	Pasadena, TX	Yes	Yes
Georgia Pacific	Plaquemine, LA	Yes	No
Hooker Chem & Plastic	Taft, LA	Yes	Yes
Jefferson Chem.	Port Neches, TX	Yes	No
Jones Chem.	Houston, TX	Yes	No
Kaiser Aluminum	Gramercy, LA	Yes	Yes
Olin Corp.	Westlake, LA	No	-
PPG Industries	Corpus Christi, TX	Yes	No
	Westlake, LA	Yes	Yes
Regent Chem. & Reas.	Port Arthur, TX	No	-
SEC Corp.	Albuquerque, NM	No	-
Shell Oil Co.	Deer Park, TX	Yes	No

Stauffer Chem.	Westlake, LA	No
Texas Analine Dye Co.	Hempstead, TX	No
Vulcan Materials	Denver City, TX	No

* Listed Elsewhere

TABLE III

HAND PRESSED AND BLOWN GLASS PLANTS

COMPANY	LOCATION	NPDES PERMIT	LEAD LIMIT
Caparion Glass Co.	Stigler, OK	No	-
Carlsbad Enchanted	Carlsbad, NM	No	-
Crystal Craft Manuf.	Shawnee, OK	No	-
EMC Glass Corp.	Decatur, TX	No	-
General Steel Ind.	Paris, TX	No	-
Glenco, Scientific	Houston, TX	No	-
Houston Glass Fabricating	Houston, TX	No	-
Hycel, Inc.	Houston, TX	No	-
Kerr Glass Manuf.*	Sand Springs, OK	Yes	No
Libby Glass (Owens-Ill)*	Shreveport, LA	Yes	No
Marvin Lindsey Glassblow	San Antonio, TX	No	-
Multi Color Glass	San Antonio, TX	No	-
Old Hickory Glass	Spiro, OK	No	-
Olszewski Stained Glass	Corpus Christi, TX	No	-
Overmyer-Perran Glass	Tulsa, OK	No	-
Owens-Illinois*	Waco, TX	Yes	No
Precision Filter Co.	Pampa, TX	No	-
Sallisaw Glass Co.	Sallisaw, OK	No	-
Scientific Glass & Instru.	Houston, TX	No	-
Scott Depot Glass Co.	Pocola, OK	No	-
Sooner Glass Manuf.	Spiro, OK	No	-
Southwestern Sci. Glass	Santa Fe, NM	No	-
Stephens Farms	Green Forrest, AR	No	-
Thomas Industries	Fort Smith, AR	No	-

Trademaster	Arlington, TX	No
United Glass & Mirror	Dallas, TX	No

* Listed Elsewhere

TABLE IV

PRIMARY AND SECONDARY STORAGE BATTERY MANUFACTURING FACILITIES

COMPANY	LOCATION	NPDES PERMIT	LEAD LIMIT
Ace Battery	Fort Worth, TX	No	-
Albuquerque Battery	Albuquerque, NM	No	-
Auto Battery & Electric	Tulsa, OK	No	-
Continental Battery	Dallas, TX	No	-
Cresent Battery & Light	New Orleans, LA	No	-
Daniell Battery Manuf.*	Baton Rouge, LA	No	-
Eagle Battery Factory	El Paso, TX	No	-
ESB, Inc.*	Dallas, TX	No	-
Ewing Battery	Brownsville, TX	No	-
General Battery Co.*	Dallas, TX	No	-
Globe Union*	Garland, TX	No	-
Gould, Inc.*	Shreveport, LA	Yes	Yes
Green Manuf. Co.	Dallas, TX	No	-
Houston Battery	Houston, TX	No	-
Hubbard Corp.	Albuquerque, NM	No	-
Hy-Vi Battery Rebuilder	Tulsa, OK	No	-
Marathon Battery	Waco, TX	No	-
Mor-CO Battery	Albuquerque, NM	No	-
Prestolite Battery*	Oklahoma, OK	No	-
Red Diamond	Hot Springs, AR	Yes	Yes
Republic Battery	Houston, TX	No	-
Standard Electric*	San Antonio, TX	No	-
Texford Battery Co.	Houston, TX	No	-

Valley Battery Co.	McAllen, TX	No

* Listed Elsewhere

TABLE V LOUISIANA - AIR LEAD SOURCES

COMPANY	LOCATION	AIR EMISSION 1976 Tons/Yr.
American Can	New Orleans	0.3 lbs/yr
Bogalusa Iron	Bogalusa	0.38
Daniell Battery	Baton Rouge	1.86
Dixie Metals	Helfin	1.82
Ethyl Corp.	Baton Rouge	107.1
Gould Battery	Shreveport	1.57
Lone Star	New Orleans	2.32
Louisiana Cement*	New Orleans	47.84*
Pearce Foundry	Prairieville	0.33
Schuylkill	Baton Rouge	22.0
Vicon Asphalt	?	108.0 lb/yr
Vulcan	Denham Springs	3.6

* Louisiana Cement uses an electro static precipatator which reduces its emissions by greater than 90%. Therefore, actual emission is less than 4.8 tons/yr.

TABLE VI TEXAS - AIR LEAD SOURCES

COMPANY	LOCATION	AIR EMISSION 1978 TONS/Yr
ASARCO, Inc.	El Paso	82
Dixie Metal	Dallas	52
ESB, Inc.	Dallas	7
Ethyl Corp.	Pasadena	61 (292)*
Gould, Inc.	Frisco	40
Houston Lead	Houston	34
Lone Star Steel	Lone Star	60
Nalco Chem.	Freeport	10
PPG Ind.	Beaumont	9 (374)
RSR Corp.	Dallas	24
Tyler Pipe	Tyler	52

* ( ) total organic and inorganic

APPENDIX A

Lead Manufacturing, Production, Usage and Emission Sites (NPDES Permit noted).

Arkansas

Hot Springs

Red Diamond Battery -permit AR0001040

Lead Bottoms (lead sulfate) CAS No. 7446-14-2
100 thousand - 1 million lbs.*
*1977 Annual production data given when available

Lonoke

Remington Arms -permit AR0001163

2,4,6,-Trinitro-1,3-Benzenediol, lead (2+) salt 10-100 thousand lbs. 15245-44-0

Louisiana

Baton Rouge

Daniel Battery Mfg. Co. -no permit

Lead Bottoms (lead sulfate) 7446-14-2
1-10 million lbs.

Lead Oxide 1309-60-0
1-10 million lbs.

~~Ethyl Corp.~~ -permit LA0004090

Lead 7439-92-1

Sodium alloy, nonbase, Na, Pb 12740-44-2

Tetraethyl lead 78-00-2

Tetramethyl lead 75-74-1

Schuylkill Metals -permit LA0004464

Lead emissions

Bogalusa

Bogalusa Iron -no permit

Lead emissions

Denham Springs

Vulcan Foundary

lead emissions

Geismar

Vulcan -permits LA0002933 & LA0034584

lead emissions

Helfin

Dixie Metals

lead emissions

Lake Charles

Cities Services-butyl plant

-permit LA0005941

Disodium plumbate 12034-30-9

New Orleans

American Can Co. -permit LA0003891

lead emissions

Lone Star Industries -permit LA0002976

lead emissions

Louisiana Cement -no permits

lead emissions

Owens-Illinois - plant 08 -permit LA0005819

Glass Oxide (lead borosilicate) 65997-17-3
100-500 million lbs.

Prairieville

Pearce Foundry -no permit

lead emissions

Shreveport

Gould, Inc. -permit LA0005657

lead bottoms (lead sulfate) 7446-14-2
1-10 million lbs.

Owens-Illinois - plant 28 - no permit

Glass Oxide (lead borosilicate) 65997-17-3

Unknown

Vicon Asphalt

lead emission

New Mexico

Grant

Gold Star No. 3 mine

lead emission

Hurley

Ground Hog Mine

lead mining - 1931 tons

Oklahoma

Ada

Brookway Glass Co. Plant 14 -permit OK0001546

Glass Oxide (lead borosilicate) 65997-17-3

Bartlesville

National Zinc -permit OK0000345

Lead residues 69012-70-0

10-500 million lbs

Henryetta

Midland Glass -no permit

Glass Oxide (lead borosilicate) 65997-17-3

Jones

Madewell and Madewell -no permit

Lead reclamation

Muskogee		
Brockway Glass - plant 3	-no permit	
Glass Oxide (lead borosilicate)		65997-17-3
Corning Glass Works	-no permit	
Glass Oxide (lead borosilicate) 10-50 million lbs.		65997-17-3
Yaffee Iron and Metal	-no permit	
Lead scrap dealer		
Oklahoma City		
Presolite Co.	-no permit	
Lead		7439-92-1
Lead Oxide		1309-60-0
Okmulgee		
Ball Glass Container Corp	-no permit	
Glass Oxide (lead borosilicate) 100-500 million lbs.		65997-17-3
Sand Springs		
~~Kerr Glass Manufacturing Co.~~	~~-no permit~~	
Glass oxide (lead borosilicate) 100-500 million lbs		65997-17-3
Sapulpa		
Liberty Glass Co.	-no permit	
Glass oxide (lead borosilicate)		65997-17-3
Tulsa		
Pennwalt Corp.	-no permit	
Lead fluoride more than 100 thousand lbs.		7783-46-2

Texas		
Amarillo		
ASARCO - closed -		
Lead slag 100 thousand - 1 million lbs.		69029-71-6
Beaumont		
PPG Ind.	-permit TX005302	
Diethyl dimethyl lead 10-50 million lbs.		1762-27-2
Ethyltrimethyl lead 1-10 million lbs.		1762-27-2
Sodium alloy, nonbase, Na, Pb 100-500 million lbs.		12740-44-2
Tetraethyl lead 50-100 million lbs.		78-00-2
Triethylmethyl lead 10-50 million lbs.		1762-28-3
Corsicana		
Chattanooga Glass Co.	-no permit	
Glass oxide (lead borosilicate) 100-500 million lbs.		65997-17-3
U. S. Gypsum Co.	-permit TX0007056	
Glass oxide (lead borosilicate) 50-100 million lbs.		65997-17-3
Corpus Christi		
ASARCO	-permit TX0003191	
Zinc Smelter - lead emissions		

Dallas		
ASARCO	-no permit	
lead emissions		
Dixie Industries, Inc.	-no permit	
lead emissions		
ESB, Inc.	-no permit	
lead oxide		1309-60-0
lead oxide		1317-36-8
General Battery Corp	-no permit	
lead bottoms (lead sulfate) 1-10 million lbs.		7446-14-2
lead oxide 10-50 million lbs.		1317-36-8
Gould Inc.	-no permit	
lead bottoms (lead sulfate) 1-10 million lbs		7446-14-2
Murdock Lead Co.	-no permit	
lead emissions		
N. L. Ind.	-no permit	
lead 10-50 million lbs.		7439-92-1
lead oxide 10-50 million		1317-36-8
RSR	-no permit	
lead emissions		
Southern Lead	-no permit	
lead emissions		
El Paso		
ASARCO, Inc.	-no permit	
Lead 100-500 million lbs		7439-92-1
Lead Oxide 1-10 million lbs		1317-36-8

closed

Phelps Dodge Ref. Co.	-no permit	
Lead emissions (copper smelter)		
Freeport		
Nalco Chemical	-permit TX0008761	
Diethyldimethyl lead		1762-27-2
Ethyltrimethyl lead		1762-26-1
Tetraethyl lead		78-00-2
Tetramethyl lead		75-74-1
Triethylmethyl lead		1762-28-3
Frisco		
Gould, Inc.	-no permit	
Lead oxide		1317-36-8
Garland		
Globe Union	-no permit	
Lead bottoms (lead sulfate) 1-10 million lbs		7446-14-2
Sherwin Williams Co.	-no permit	
Lead naphthenate		61790-14-5
10-100 thousand		
Houston		
A & B Metals & Smelting	-no permit	
lead emmission		
Anchor Hocking plant 18	-permit TX0006718	
Glass oxide (lead borosilicate)		65997-17-3
Esso Eastern	-no permit	
Tetraethyl lead imports		78-00-2

? Federated Metals Corp.	-no permit	
Flue gas, lead-tin alloy 1-10 thousand lbs		69012-60-8
Lead alloy base, Sn, dross 10-100 thousand lbs.		69011-60-5
Gulf Reduction Corp.	-no permit	
Lead emissions		
Houston Fishing Tackle	-no permit	
Lead		
✓ Houston Lead Co.	-no permit	
Lead emissions		
✓ Lead Products	-no permit	
Lead scrap		
Proler International Corp.	-permit TX0065633	
Lead emissions		
San Jacinto Paint Mfg.	-no permit	
Lead oxide imports		1314-41-6
Sterling Type, Rule & Metals	-no permit	
Lead type		
Lone Star Steel	-permit TX0000027	
Lead emissions		
Pasadena		
Ethyl Corp.	-permit TX0004731	
Lead		7439-92-1
Sodium alloy, nonbase Na, Pb		12740-44-2

Tetraethyl lead 78-00-2

Port Arthur

Texaco, Inc. -permits TX0005835 & TX0005843

Lead Napthenate 61790-14-5

100 thousand - 1 million lbs

San Antonio

Standard Electric Co., Inc. -no permit

Lead bottoms (lead sulfate) 7446-14-2

1-10 million lbs

Lead oxide 1317-36-8

1-10 million lbs

Tarrant County

National Metal & Smelting -no permit

Lead emissions

Terrell

Powerlab, Inc. -no permit

Lead oxide

Tyler

Tyler Pipe -permit TX0001465

Lead emissions

Waco

Owens-Illinois - plant 15 -permit TX0003158

Glass oxide (lead borosilicate) 65997-17-3

500 million - 1 billion lbs

Waxahachie

Kerr Glass Manuf. Co. -no permit

Glass oxide (lead borosilicate) 65997-17-3

100-500 million lbs

Wichita Falls

Certainteed Corp.

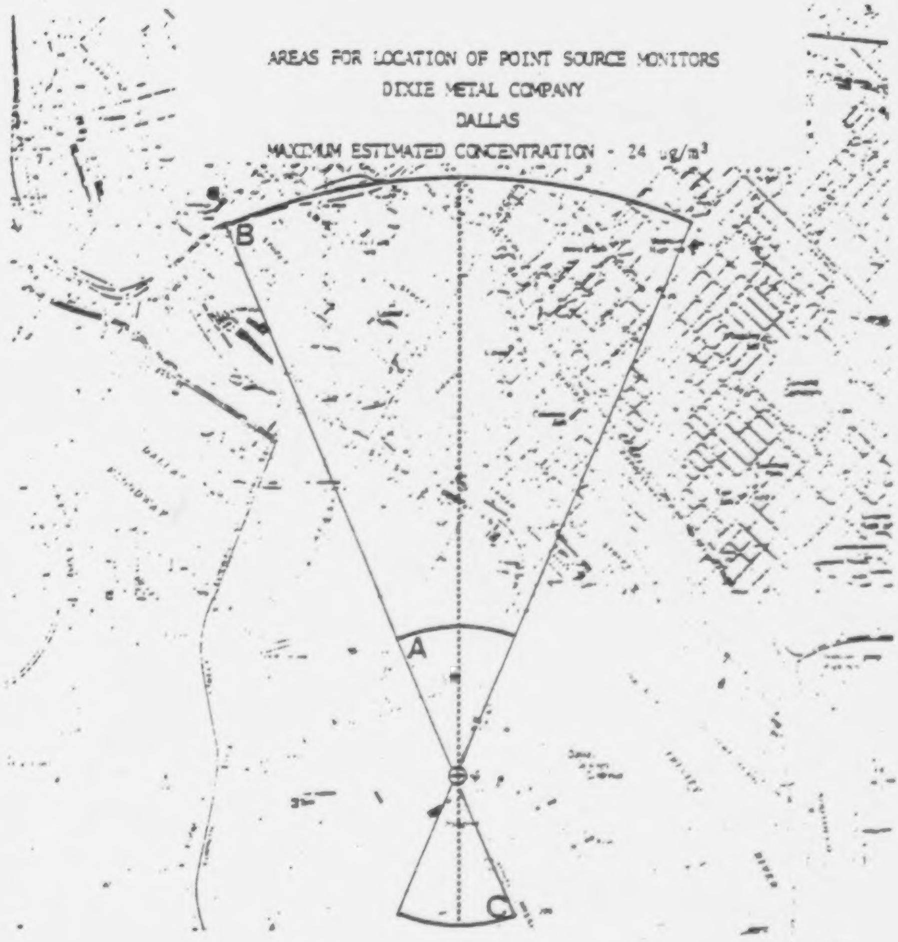
Glass oxide (lead borosilicate) 65997-17-3

1-10 million lbs

APPENDIX B

AREAS FOR LOCATION OF POINT SOURCE MONITORS IN TEXAS

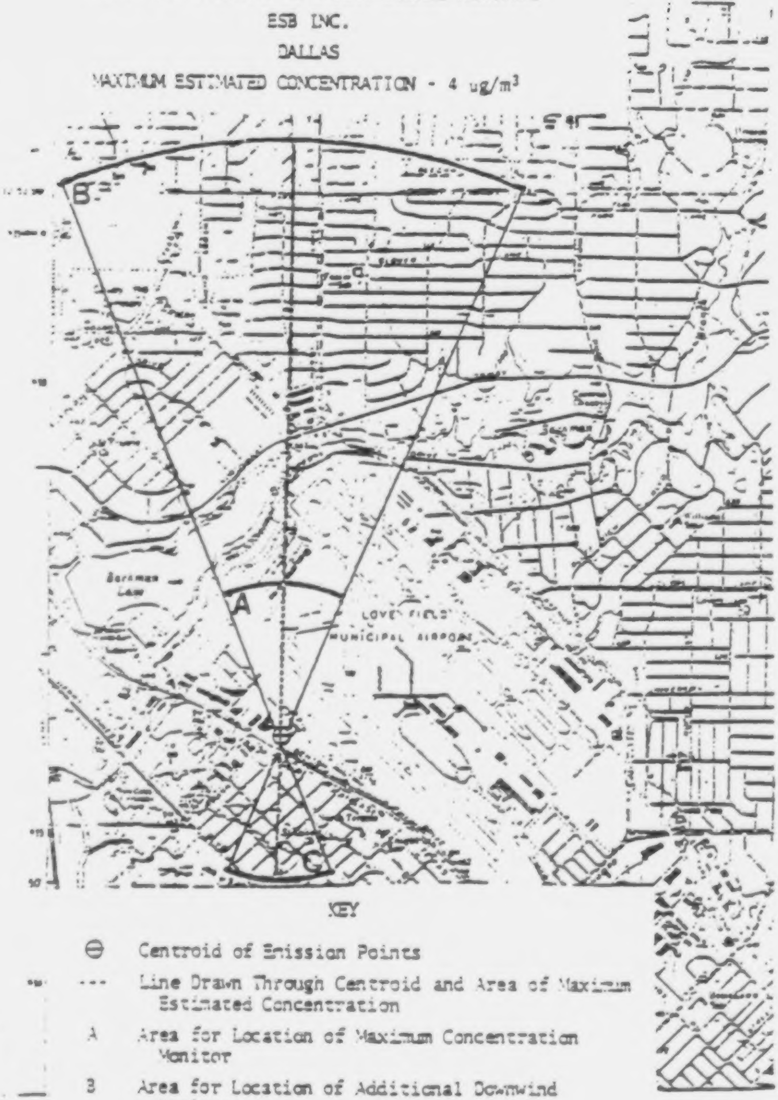
AREAS FOR LOCATION OF POINT SOURCE MONITORS
DIXIE METAL COMPANY
DALLAS
MAXIMUM ESTIMATED CONCENTRATION - 24 ug/m³



KEY

⊖	Centroid of Emission Points
---	Line Drawn Through Centroid and Area of Maximum Estimated Concentration
A	Area for Location of Maximum Concentration Monitor
B	Area for Location of Additional Downwind Monitor
C	Area for Location of Upwind Monitor

AREAS FOR LOCATION OF POINT SOURCE MONITORS
ESB INC.
DALLAS
MAXIMUM ESTIMATED CONCENTRATION - 4 ug/m³


B
A
C
Bachman Lake
LOVE FIELD
MUNICIPAL AIRPORT

KEY

⊖ Centroid of Emission Points

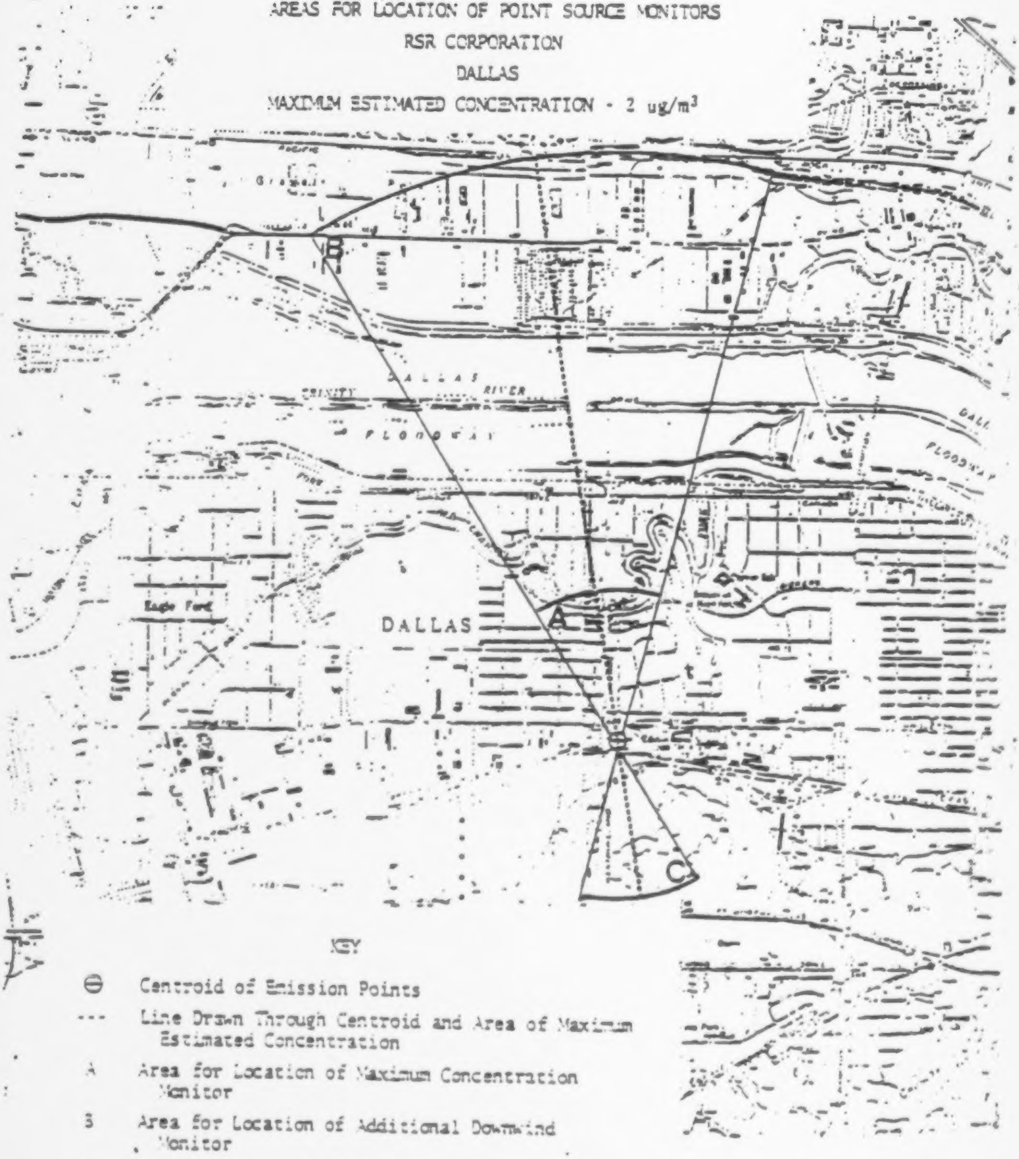
--- Line Drawn Through Centroid and Area of Maximum Estimated Concentration

A Area for Location of Maximum Concentration Monitor

B Area for Location of Additional Downwind Monitor

C Area for Location of Upwind Monitor

AREAS FOR LOCATION OF POINT SOURCE MONITORS
RSR CORPORATION
DALLAS
MAXIMUM ESTIMATED CONCENTRATION - 2 ug/m^3

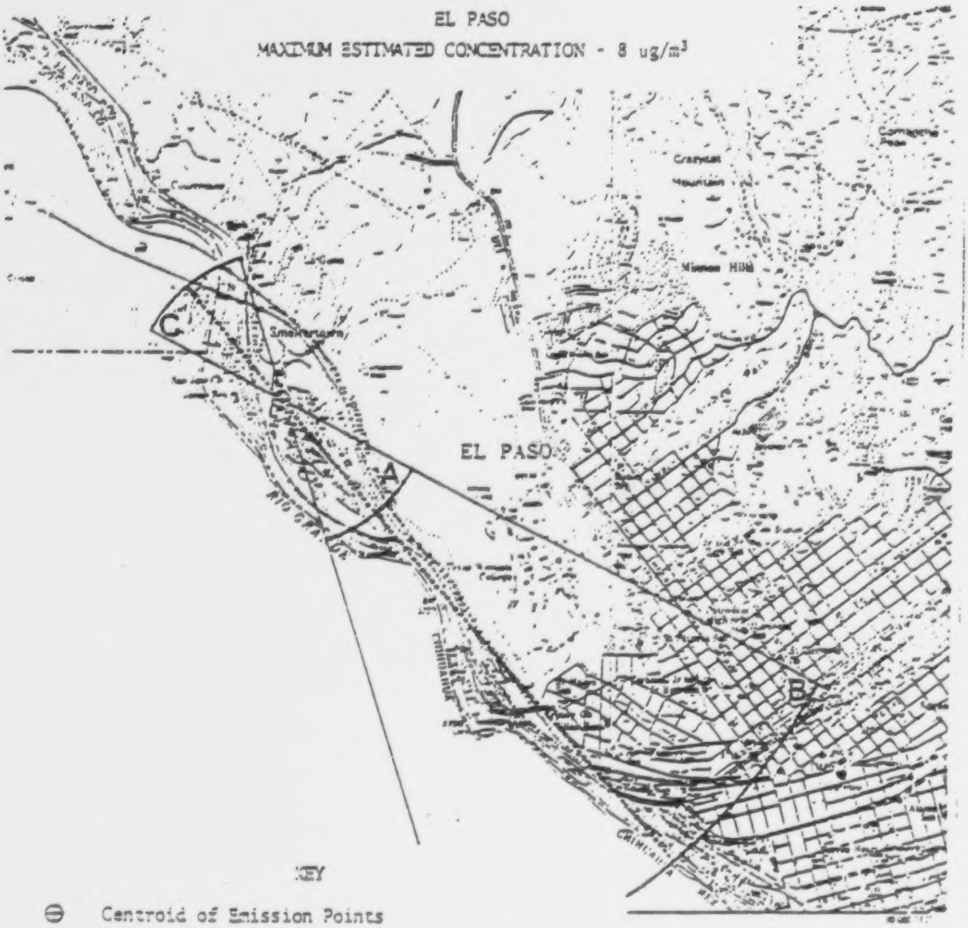


B
DALLAS
TRINITY
RIVER
FLOODWAY
DALLAS FLOODWAY
Eagle Ford
DALLAS
A
C

KEY

⊖	Centroid of Emission Points
---	Line Drawn Through Centroid and Area of Maximum Estimated Concentration
A	Area for Location of Maximum Concentration Monitor
B	Area for Location of Additional Downwind Monitor
C	Area for Location of Upwind Monitor

AREAS FOR LOCATION OF POINT SOURCE MONITORS
ASARCO
EL PASO
MAXIMUM ESTIMATED CONCENTRATION - 8 ug/m^3


C
A
B
EL PASO
Smeltertown
Crazycat Mountain
Mission Hills
RIO GRANDE

[illegible] by the Army Map Service

[illegible]

[illegible] USGS, USC&GS and USCE

KEY

⊖ Centroid of Emission Points

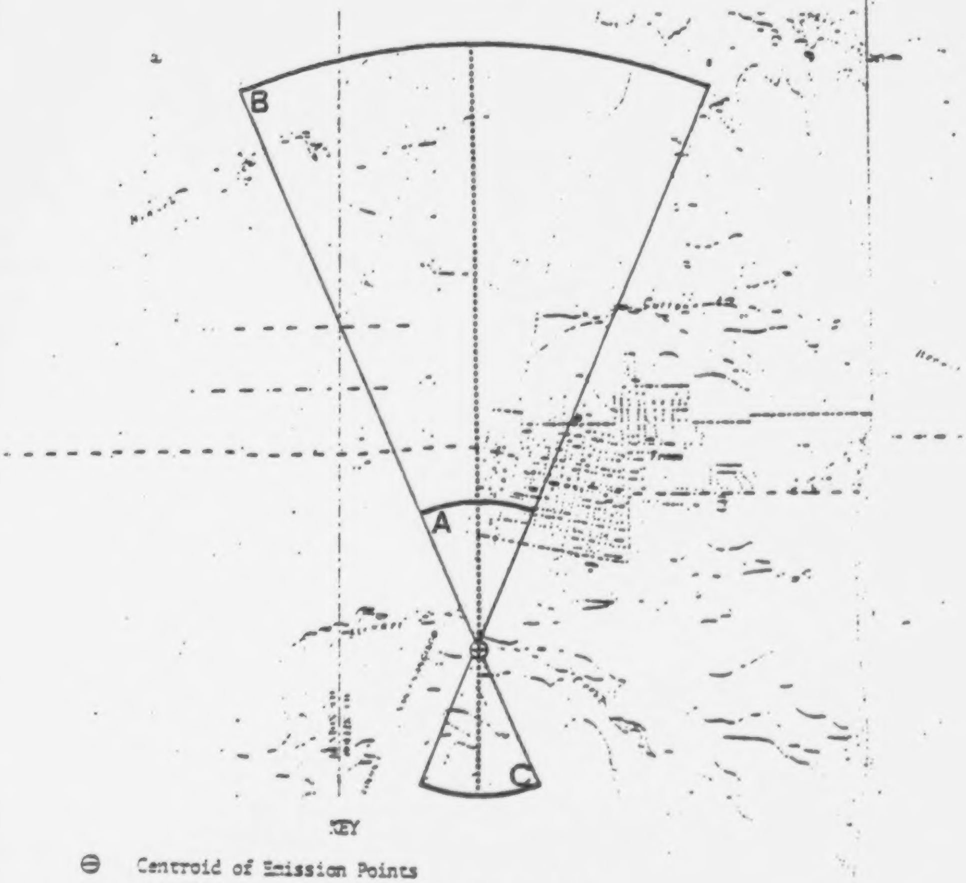
--- Line Drawn Through Centroid and Area of Maximum Estimated Concentration

A Area for Location of Maximum Concentration Monitor

B Area for Location of Additional Downwind Monitor

C Area for Location of Upwind Monitor

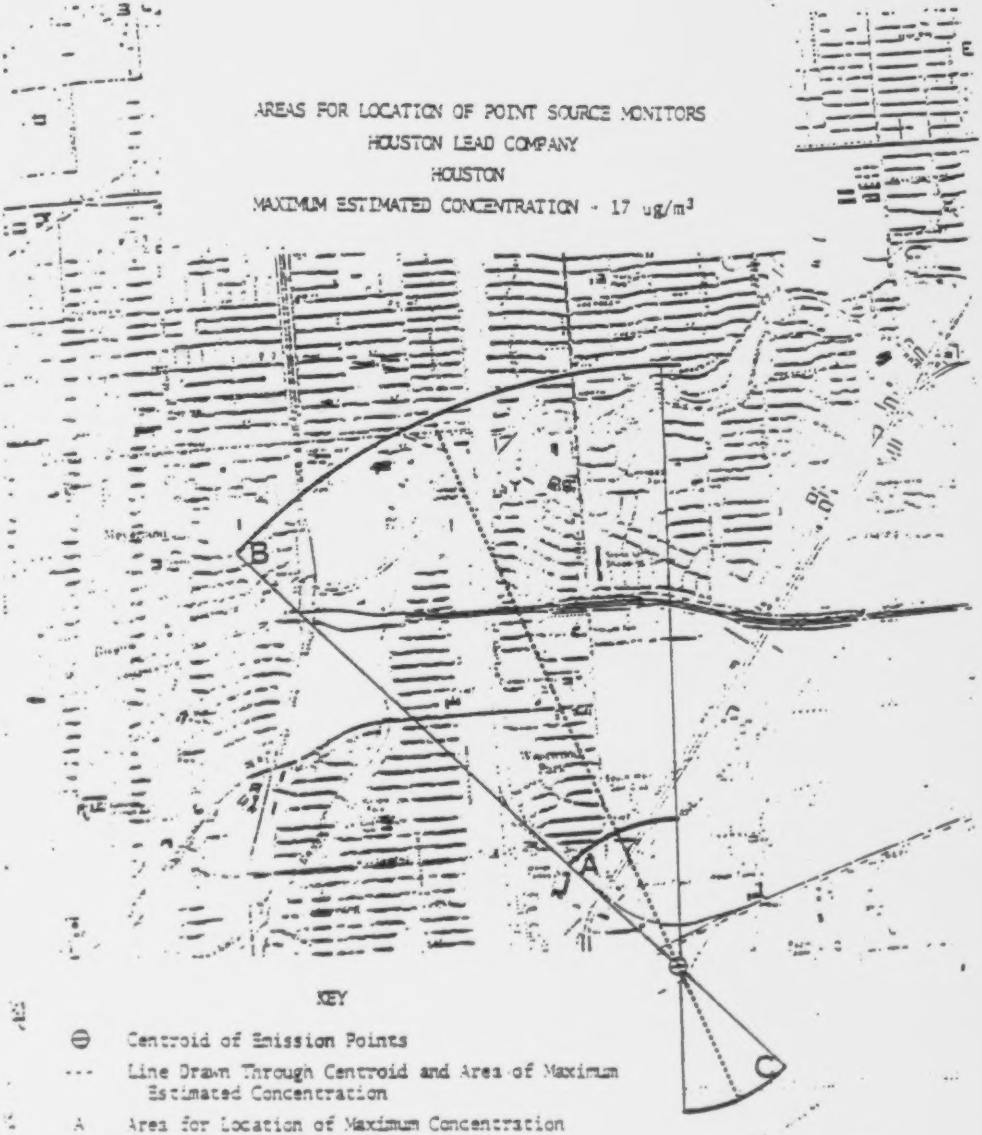
AREAS FOR LOCATION OF POINT SOURCE MONITORS
GOULD INC.
FRISCO
MAXIMUM ESTIMATED CONCENTRATION - 12 µg/m³



KEY

⊖	Centroid of Emission Points
---	Line Drawn Through Centroid and Area of Maximum Estimated Concentration
A	Area for Location of Maximum Concentration Monitor
B	Area for Location of Additional Downwind Monitor
C	Area for Location of Upwind Monitor

AREAS FOR LOCATION OF POINT SOURCE MONITORS
HOUSTON LEAD COMPANY
HOUSTON
MAXIMUM ESTIMATED CONCENTRATION - 17 $\mu g/m^3$



KEY

⊖	Centroid of Emission Points
---	Line Drawn Through Centroid and Area of Maximum Estimated Concentration
A	Area for Location of Maximum Concentration Monitor
B	Area for Location of Additional Downwind Monitor
C	Area for Location of Upwind Monitor

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